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MEDICAL AND SURGICAL REPORTS

OF

THE BOSTON CITY HOSPITAL.

Thirteenth Series.

EDITED BY

HERBERT L. BURRELL, M.D., W. T. COUNCILMAN, M.D.,
AND CHARLES F. WITHINGTON, M.D.

BOSTON:
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1902.

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PREFACE.

The report is now established as an annual publication, the manuscript for it being due on October 1st of each year, and with the co-operation of the contributors the editors hope to be able to issue the report on December 1st of each year.

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The Editors.

December 1, 1902.

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I.

CONGENITAL ANOMALIES OF THE PHALANGES, WITH REPORT OF CASES STUDIED BY SKIAGRAPHY.

By F. B. Lund, M.D., Assistant Visiting Surgeon.

Anomalies of the Phalanges, on account of their rarity, and on account of the fact that their possessors apply for surgical treatment, are not without interest to the surgeon. The complicated structure of the hand, which fits it so admirably for the performance of the delicate and complicated, under the influence of the will, at the same time renders it a difficult field for surgical work. When so complicated a mechanism is deranged by accident or disease, and the surgeon is required to perform operations of more or less intricacy upon it, he must use his utmost skill to avoid adhesions, injuries to nerves, tendons, or joints which will prove more or less disabling. Asepsis has fortunately almost eliminated the danger from infection of the tendon sheaths, but the mechanical difficulties presented by this class of cases still remain sufficient to tax his ingenuity. These cases too frequently remind us of the shortcomings in minor matters of our art, which in larger directions has progressed so far, and gives us results of such brilliancy. In the following paper the writer has not attempted to give a complete discussion or even enumeration of all the congenital deformities of the hand, but merely to mention such reflections as to their hereditary nature, causation and treatment as have been suggested by the cases described below, all of which have presented themselves at the City Hospital Clinic for treatment.

As these anomalies are congenital in type, the writer was led to investigate as far as possible the development of the limbs, only to find that the morphological value of vertebrate limbs had long been the subject of discussion and speculation, and that at the present time the solution of the problem is theoretical rather than positive. (Minot. Human Embryology, p. 448.) The theory adopted by Minot as the only one having any standing is Balfour's opinion that the limbs are specialized portions of a lateral fin-fold, similar to the dorsal and ventral median fin-folds of fishes. "From the manner of their development it is obvious that the limbs have a flattened form and a dorsal (or extensor) surface, and a ventral (or flexor) surface, and as soon as they project from the body, as they do at right angles, there is an anterior or cranial border, and a posterior or dorsal border." The ridge forming the rudiment of each fin-fold becomes broader, and its attachment to the side of the body becomes shortened from behind forward, so that what was originally the attached border becomes the posterior border. The primitive skeleton of the fins consists of a longitudinal bar running along the base of the fin, and giving off at right angles a series of rays which pass into the fin. Balfour's observations show that there was a primitive longitudinal skeletal piece at the base of the limb fold, and from this rays are developed which run out into the fold. In the higher fishes these rays come off on either side of the skeletal limb axis, instead of as in the lower fishes only on the cranial side, forming the archipterygium, which is regarded as the ancestral form of the pentadactyl limbs in amphibia and mammals.

The Cheiropterygium is the arch type of the pentadactyle-limb, and its essential characteristic is the division into four segments, as follows (Minot):

1. { Upper Arm. / Upper Leg. } 2. { Forearm. / Lower Leg. } 3. { Carpus. / Tarsus. } 4. { Hand. / Foot. }

The upper segment contains one long bone, the humerus or femur, the second two long bones (radius or tibia and ulna or fibula), the third segment nine small bones, carpals or tarsals, and the fourth consists of separate digits, five in number,

FIGURE 1.

each with its metacarpal bone. Into the homologies of the human carpus and phalanges with the typical vertebrate form it is not necessary to enter here. It is sufficient to say that there is one series of bones which represents the true axis of the limb, and the other bones represent a series of rays coming off from it. This supposed axis in the arm begins with the humerus and is carried through the ulna,

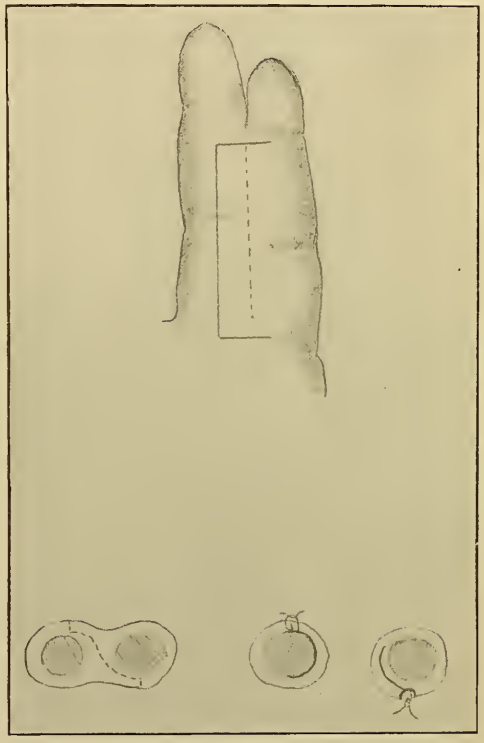

FIGURE 2.

and terminates with one of the digits, with which digit authorities are not agreed, nor is it necessary for the purposes of our study for the writer to attempt to decide.

"The skeleton of the arm in mammals, in its earliest mesenchymal stages, forms an uninterrupted anlage, with no indication of its future subdivision, and is moreover prob-

ably continuous with the anlage of the pectoral girdle." (Minot.) As soon as ossification begins the individual skeletal pieces are indicated by separate centres of ossification, which begin near the centre of each piece and spread toward the periphery. The separation of each digital series is shown in the primitive mesenchymal anlage, which also

FIGURE 3.

shows traces of a sixth digit in front of the thumb, which normally persists as a rudiment and only for a short time. It is undoubtedly the occasional persistence of this sixth digit which accounts for the most frequent digital deformity which is presented to the surgeon, namely, the double thumb.

This condition is shown in the radiograph (Fig. 1). The extra thumb is commonly not completely separated from the thumb proper, and the bones are more or less fused with those of the normal member. The condensed mesenchyma between two adjacent cartilages becomes fibrillar, and thus the joints are produced.

In the human embryo at six weeks nearly all the skeletal pieces are present, and by the eighth week ossification has begun in the humerus, radius and ulna. The carpus is

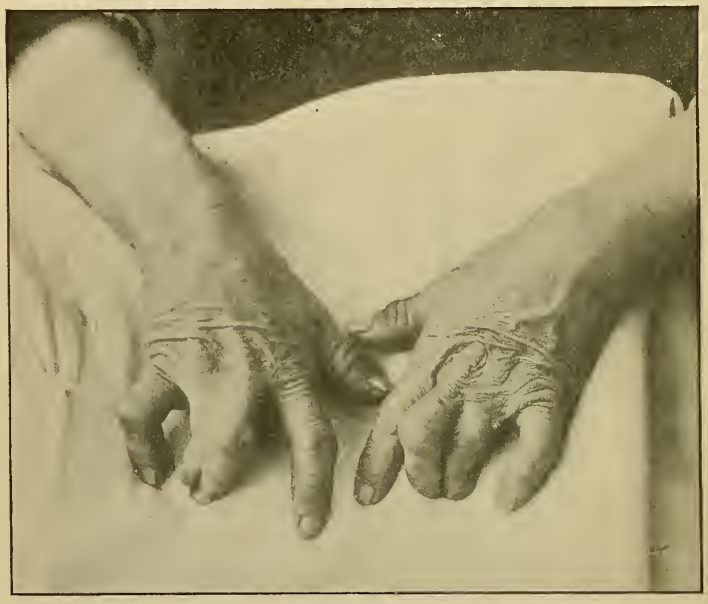

FIGURE 4.

entirely cartilaginous at birth. Ossification in the metacarpal bones and phalanges begins about the eighth week.

As the time of ossification of the various bones of the arm and hand can be found to give no aid in an attempt to elucidate the different deformities it is not here in place, and the interested reader is referred to Minot's embryology and Quain's Anatomy.

The different deformities of the hands are classified under three heads. ("*Nichols*, in Reference Hand Book of Medical Sciences. Vol. IV. 1902.")

1. Those which depend on deficient development. Acheiria (absence of the hand), ectrodactylism (absence of one or more digits), hypophalangism (absence of one or more phalanges).

FIGURE 5.

2. Excessive development. Polycheiria, polydactylism, polyphalangism.

3. Perverted development. Syndactylism, cleft hand, congenital constrictions, deflections, congenital dislocations, congenital neoplasms.

The coexistence of different types is common, such for instance as the combination of syndactylism with anomalies and deficiencies of the phalanges, which is the type illustrated by the first group of cases to be presented in this

FIGURE 6.

paper. The coexistence of anomalies and deficiencies of other parts of the body with deformities of the phalanges is common. Harelip and cleft palate are not infrequent in patients with deformed phalanges. In Case III., here reported, the coexistence of syndactylism with deformity of

the phalanges, and syndactylism with supernumerary toes in the same patient is noted. Hereditary transmission is common, and a tendency to the reversion to the same or analagous deformities in families is exemplified in the two families which form the basis of this paper.

The causes are obscure, and in cases such as those here reported, can only be assigned to "deep-seated innate tendencies to aberrant development, often definite and fixed, and

FIGURE 7.

hereditary, the same deformity appearing in different generations." — *Nichols. Loc. cit.* Intra-uterine ulcerations and adhesions, and other pathological processes may for the purpose of such deformities as are here presented in this paper be left out of account, as the deformities involve not only the skin and subcutaneous tissues, but also the bones, and undoubtedly the muscular tissues, etc., as well.

Referring to the evolutionary and embryological outline of the development of the hand as given above, it becomes evi-

dent that the arm and hand are homologous with the pectoral fin of fishes, the wing of the bird and bat, and the fore

FIGURE 8.

limb of mammals. It is an interesting matter for speculation whether the webbing of the fingers may not be a reversion to some ancestral type in which the digits were

united by a web. The fact that in all the cases which have come under the writer's observation syndactylism has been associated with anomalies of the phalanges and sometimes

FIGURE 9.

of the metacarpal bones, points to developmental origin of the deformity, and at the same time has a practical bearing as increasing the difficulty of treatment. In these cases the

fingers are not only united by a web, but the soft parts of the adjacent fingers are practically one, forming a band of union as thick as the fingers themselves, and even the bones themselves are frequently united, so that even if the fingers are separated it becomes practically impossible to get skin

FIGURE 10.

flaps with which to cover the adjacent raw surfaces. Operations for discission and suture, with or without the preliminary formation of a fistula at the base of the fingers, are obviously applicable only to cases in which the digits are united by a comparatively thin flap. The only method

applicable to fingers united by thick flaps, the majority of operable cases, is the method known as Didot's operation, in which a dorsal flap is taken from one finger and a palmar

FIGURE II.

flap (Fig. 2) from the other. Certain difficulties connected with this operation will be mentioned in connection with Case III.

CASES OF SYNDACTYLISM WITH BONY DEFORMITIES OF THE PHALANGES.

A case of web fingers associated with anomalies of the phalanges which the writer considered inoperable was reported in the Boston Medical and Surgical Journal of February 18, 1897, and the skiagram is

FIGURE 12.

here reproduced (Fig. 3). The patient was a girl of eighteen years of age in whom the fingers were so closely united that the hand resembled the flipper of a seal. As will be seen by reference to the cut, the bones of the last three fingers were so close together that it would have been impossible to separate them and get skin flap enough to cover the sides of the phalanges. The anomalies presented, as will be seen by reference

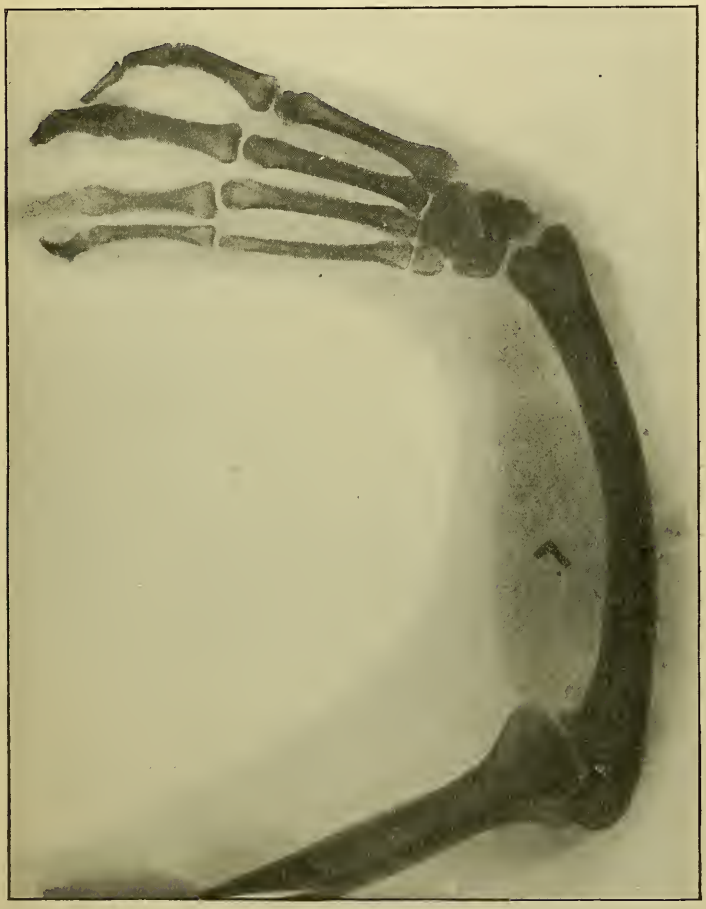

FIGURE 13.

to the figure are in the absence of the second phalanx of the forefinger, and reduplication of the last phalanx, which is united by a bony bridge to the terminal phalanx of the middle finger. The middle finger shows a rudimentary metacarpal bone, in the ring finger the second phalanx is absent, and in the little finger it is rudimentary. The single nail extending across the whole width of the hand represents the union of the four nails.

The four cases about to be reported are of that of an Italian, forty-five years of age, and three of his children.

Case I. — The hands of Case I. are shown in Fig. 4, in a skiagram of his right hand in Fig. 5, and his left hand in Fig. 6. An attempt had been made when he was a child at a hospital in Italy to separate the middle and ring fingers of his left hand. As will be seen by reference to the figure this was only partially successful, and the reason for the lack of success may be seen in the bony deformities shown in these skiagrams. At present the fingers of both hands are more or less permanently flexed, the second and third phalanges being incapable of extension much beyond a right angle with the plane of the hand. His business which is that of driving a fruit wagon is considerably interfered with by the stiffness of his deformed fingers and the clumsiness of the motions of his hands.

Case II. — Is that of the daughter of Case I. Her hands are represented in Fig. 7, a skiagram of her right hand is shown in Fig. 8, and of her left hand in Fig. 9. When she was an infant an attempt was made to separate the middle and ring fingers of the right hand at the Boston City Hospital.

As will be seen by reference to the skiagram the first phalanx of the ring finger of the right hand is shortened and very much deformed, so that its articular surface faces outward instead of directly upward. The web between the two fingers, which was complete, has been separated as a result of the operation down to the level of the second joint of the ring finger, but the ring finger cannot be extended beyond about forty-five degrees, and is held in a position crossing the ventral surface of the middle finger.

The ring and middle fingers of the left hand as will be seen by reference to the photograph, are firmly united, while there is an attempt at reduplication of the two terminal phalanges of the ring finger. Between the heads of the metacarpals are several irregular bones which may represent additional rudimentary metacarpals or phalanges, but the homologies of which it is impossible to make out accurately. Operative relief in this case is clearly out of the question.

Case III.— Is a female child, sister of Case II., aged three years, who presented a union of the ring and middle fingers of both hands by a thick web, together with an outward deviation of the terminal phalanges of both ring fingers similar to that presented in the right hand of Case II. This child also had two supernumerary digits on each foot which were united to the fifth toes by a web and to each other also. Didot's operation for the separation of the phalanges was performed upon the right hand by the writer and upon the left by Dr. Munro. Inasmuch as the middle finger of both hands was the larger, stronger and more normal in appearance of the two which were united, the palmar flap was taken from the ring finger and the dorsal flap from the middle finger, so that when the operation was completed the longitudinal scar was upon the dorsal surface of the middle finger and the palmar surface of the ring finger. The result six months later showed that by the operation separation was accomplished down nearly to the normal position of the web of the fingers, but upon both hands, espe-

FIGURE 14,

cially upon the one operated on by the writer, contraction of the scar had caused more or less permanent flexion of the ring fingers. This contraction may perhaps be ordinarily avoided in favorable cases by bringing the scar to the side of the fingers, but in this case the fingers were so closely united that it was impossible to obtain sufficient flap to do this, and the result illustrates another of the difficulties presented by cases of this class. The child could not be induced to keep her hands still long enough to have a skiagram or photograph taken.

Case IV. — Was that of an infant brother of the two preceding children, aged nine months. He also had a union of the middle and ring fingers of both hands. This child also could not be induced to sit for his photograph. The mother of these children was healthy and had normal fingers and toes. No further family history could be obtained from the patients, who talked no English.

Case V. — Is that of a laborer, seventy years old, a native of Ireland. He could give no information as to his family history. His right hand was normal. His left hand is shown in Fig. 10, and a skiagram is seen in Fig. 11. He had been a sailor the greater part of his life, and when he was about thirty-five years of age an attempt was made by a surgeon in Ireland to separate his ring and middle fingers, which were united by a web. The attempt was not successful, the fingers remaining about as before. Within the last four or five years both fingers had begun to enlarge until they had assumed the irregular shape shown in the photograph and skiagram. Palpation showed that they presented hard, irregular masses suggestive of osteo-chondroma, and this diagnosis is confirmed by the skiagram, which shows the irregular enlargement of the bones, which were undoubtedly deformed before the abnormal growth took place. They are interesting as showing that deformed phalanges may become the seat of osteo-chondroma.

CASES OF HEREDITARY DEFORMITIES OF MIXED TYPE.

Case VI. — A case of absence of the radius and thumb associated with lack of development of the corresponding humerus, clavicle and scapula. This patient's photograph is shown in Fig. 12, the skiagram of the forearm in Fig. 13, and the shoulder in Fig. 14. The patient was a young woman seventeen years old whose family history was kindly furnished by my colleague, Dr. Horace Arnold. Four brothers living in Nova Scotia were all married and had children, more or less of whom were deformed.

Jonathan H. had eight children, one of whom, Celia S., the mother of Case VI., had a supernumerary phalanx in her thumb. Her hand is shown in Fig. 15.

The second brother, Henry H., had several children, two of whom, one son and one daughter, were deformed.

The third brother, Nathan H., had several children, one of whom, a son, was deformed.

The fourth brother, Thomas H., had several children, three of his daughters and two of his sons being deformed. Of this family one of the daughters had no arms, merely short appendages to which four fingers each were attached.

FIGURE 15.

Case VI. was the granddaughter of Jonathan H. The only deformity presented by her mother (Case VII.) was a supernumerary phalanx in the thumb of the right hand. The deformities in Case VI., as will be seen by reference to the skiagram, consist of absence of the radius and thumb and probably of two of the carpal bones, although the condition of the latter cannot be accurately determined by the skiagram (Fig. 13), as they lie so closely together that their boundaries cannot be made out. Associated with this is curious lack of development of the entire upper portion of the body, the ulna being curved and shortened, and the humerus, scapula and clavicle being under developed, as will be seen by reference to Fig. 12. The head of the humerus in particular appears under developed (Fig. 14), and fills out the glenoid cavity to such a slight extent as to suggest a congenital dislocation. The acromion process is curiously curved downward over the undeveloped head of the humerus, and it, as well as the clavicle, is very much smaller than those of the opposite side, which are normal in every particular. The skiagram of the entire thorax shows this particularly well, but there is no space to present it in this paper. The radial displacement of the hand resulting from the absence of the radius gives it the position known as the "club" hand. The patient is able to do almost all kinds of work, and does fine sewing and fancy work well. All the motions of the fingers are apparently normal.

Case VII. — Mother of Case VI., as mentioned above and illustrated in the skiagram, Fig. 15, presents a supernumerary phalanx in the thumb.

Case VIII. — E. H., nineteen months old, grandson of Jonathan H., nephew of Case VII., and first cousin of Case VI., presents the same deformity as Case VI. He could not be induced to keep quiet long enough for a photograph or skiagram to be taken. He uses his hand to pick up things, has a firm grasp, and uses this hand to assist the other when eating or drinking.

These last three cases are interesting, from the point of view of family history, as examples of a tendency to deformity transmitted in a single family, and apparently both through the male and female line.

With regard to the treatment of such cases as Case VI., apparently nothing can be done to straighten the hand except the doubtful expedient of grafting the bone of an animal to supply the place of the absent radius and pushing the hand around into position, which newspaper reports state has been done by Dr. Roswell Park in a similar case in a child. In the cases observed in this paper the hands were so useful, in spite of the deformity, as to render any such procedure uncalled for.

For the radiographs which so well illustrate these cases the writer is indebted to Dr. F. H. Williams and Mr. Fewkes, the hospital photographer.

In conclusion, although no important results of a practical character have been secured by the radio-graphic study of web fingers, it has certainly been demonstrated that the deformity, unlike beauty, is more than skin deep, and presents problems of a most formidable character to the surgeon.

II.

THE PATHOGENESIS AND PATHOLOGICAL ANATOMY OF ENLARGED PROSTATE.

By L. R. G. Crandon, M.D.

[From the Pathological Laboratory of the Boston City Hospital.]

This work is undertaken largely with the view to investigate the conclusions of the monumental work of Ciechanowski (1 and 2). To him, more than to any other, are due the thanks of the profession for new and careful work and for deliberate and conservative conclusions.

For personal direction and help in this study I wish to give the most sincere thanks to Dr. F. B. Mallory. The surgical specimens have been very kindly given to me by Dr. Paul Thorndike, and for unlimited access to post-mortem material I am indebted to Dr. Councilman and Dr. Mallory.

The number of senile prostates examined was thirty-seven, of which twelve were enlarged, twenty-four normal in size, and one small.

Urinary symptoms, which at present may be loosely designated "prostatism," are seen in many men over fifty. The proportionate number may not be determined with any accuracy. Thompson held that there might be a slight enlargement of the prostate without symptoms in about one in three men over sixty years and that a marked enlargement is seen in one in seven after that age. The symptoms are increased frequency of micturition, incomplete emptying of the bladder, with residual urine, or distention with dribbling. Of men with these symptoms, it is estimated that only 15 per cent. have enlarged prostate, and, although it will be shown that disease and change in the bladder wall are probably important

factors in producing this condition of prostatism, nevertheless, they alone are probably not sufficient, for one never sees senile vesical inertia with retention in women, where directness and permeability of the urethra are not affected. Hence mechanical obstruction in the form of prostatic enlargement at the outlet of the bladder must be always important amongst the elements of "prostatism."

I have attempted in the following notes to compare and record differences in senile prostates, enlarged, normal, or small, and in some degree to study the accompanying bladder-wall changes.

Methods.—In general for histology, preservation in Zenker's fluid was found the best. Formalin, 10 per cent. solution, was used for hardening and preserving specimens for gross examination and also for special nerve-stains. The prostates were imbedded, some in paraffin, many in celloidin, and cut of varying thicknesses (7 to 12 mmm.). All stains were used, but principally Mayer's hemalum and eosin, Van Gieson's stain diluted and with enough additional picric acid to make the yellow predominate over the red. For minute changes in the proportions between muscle and connective tissue, Mallory's connective tissue stain (50) gives marvellous detail. Nerves and nerve-sheaths were stained by Pal's modification of Weigert's method (51). For elastic tissue Weigert's stain (52) was used.

Part I.

THE BLADDER-WALL IN VESICAL INSUFFICIENCY.

Examination in the gross of a bladder and prostate cut in a sagittal plane, through the urethra, in subjects over forty-five years, shows in over half the cases, at the origin of the urethra, a distinct lip, rising slightly above the level of the urethral floor, then dropping sharply in a perpendicular behind, to form the anterior wall of a bladder-pouch (Fig. 1). This lip is formed by the prostate, which may or may not be enlarged, and the pouch varies much in size, not corresponding necessarily to the size of the prostate. This pouch contains the residual urine, and is a prominent factor in

prostatism, and, inasmuch as it is found without enlarged prostate, we must seek a partial cause of it, at least, outside the prostate.

What changes in the bladder-wall are to be seen where this pouch and vesical insufficiency exist?

At least five possible changes are to be considered:

I. The presence of fat in the senile bladder: Dittell (17) laid the cause of senile bladder to fatty degeneration. Maas (47) laid it to fatty degeneration of hypertrophic bladder-muscles. Rokitansky (67) describes it loosely as a colloid degeneration. Ciechanowski (1, p. 207) found fat between the outer muscle bundles in streaks, occasionally subserous, more rarely submucous, where it is never normal, and he concludes that bladder insufficiency does not depend on fatty degeneration, because this fat is not seen by any means in all cases, and only rarely in muscle cells.

I have seen fat between muscle bundles and subserous, where the globules are large, and borne in cells with relatively little protoplasm; that is to say, having the characters of normal adipose tissue. Only occasionally muscle cells show fine fat globules, and then only in bundles where other and much more extensive and important changes, as will be noted, are present. With this slight exception, fat in the bladder-walls apparently has only the significance of fat deposit anywhere.

II. The presence of new connective tissue and of muscle changes in the senile bladder: Orth (61) has noticed atrophy of the bladder muscles in chronic dilatation of the bladder. Launois (43) holds that by overwork the muscle elements become surrounded with fibrous tissue, which finally suppresses their action. Guyon (32) believes that all changes of a connective tissue kind are due to the impaired nourishment consequent on arterio-sclerosis of the bladder. Bohdanovicz, quoted by Ciechanowski (1, p. 207), holds also that over-exertion of bladder muscle leads to fibrous changes, but he ignores cases of bladder insufficiency where no signs of previous hypertrophy are to be found; cases, in other words, where no cause for bladder-muscle hypertrophy exists. He gives no heed, also, to chronic inflammation as a possible

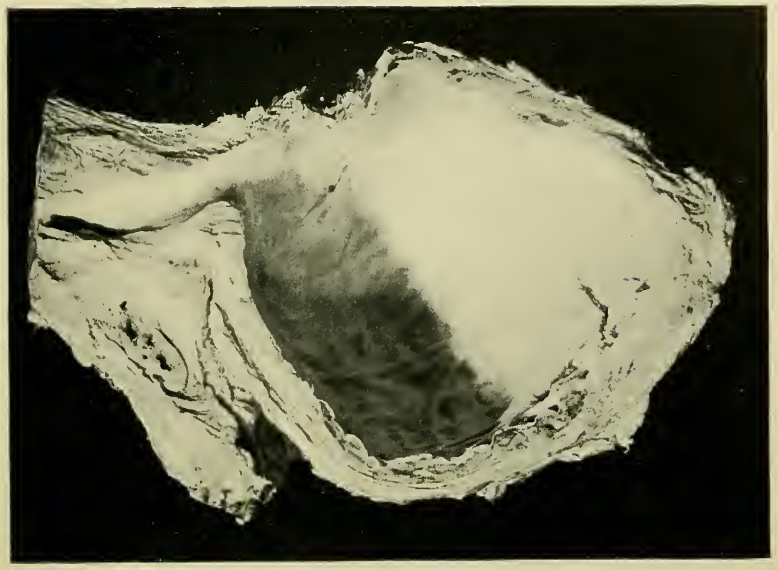

Fig. 1. Sagittal section of senile bladder and prostate, showing the fibrous bands in the wall of the atrophic bladder, the retroprostatic pouch, and the orificial prostatic lip.

Fig. 2. Showing fibrous infiltration of the bladder-wall muscles.

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cause of some of the changes which he observes. In the study of senile changes in the bladder-wall, therefore, such changes as are plainly due to chronic inflammation should be clearly distinguished.

Ciechanowski has done so, and shutting out these cases which have the submucous fibrous deposit of old cystitis, makes a series of careful measurements of transverse sections of bladder-wall. He adds together the total area of muscle elements, and compares it with the area of the interstitial connective tissue in sections of definite size, submucous and subserous tissue being excluded. He finds that the amount of connective tissue compared to that of muscle in the highest grades of arterio-sclerosis varies between 1 : 4 and 1 : 1.2. The fraction for the normal bladder given by both Orth and Ciechanowski is 1 : 3. The latter then observes that interfibrillary and interfascicular fibrous tissue only appears increased when there is a considerable amount of new connective tissue about the vessels. Hence, shutting out these cases, which from the apparent vascular origin of the fibrous tissue seem to be inflammatory in origin, he concludes that the connective tissue increase is only apparent and fills in the place of truly atrophied muscle. He compares cases which differ only in respect to age, with no cystitis, cases where no cause for bladder-muscle hypertrophy exists, and finds his relation of connective tissue to muscle go from 1 : 3.5 up to 1 : 2 according to age alone, and hence concludes that the change is true senile atrophy. Into this matter I have not gone with detail. My specimens (Fig. 2) excluding those apparently inflammatory, show irregular distribution of the new-formed fibrous tissue within the muscle bundles. Such fasciculæ of muscle as remain stain well and show striæ. The fibrous tissue between the separate muscle fibres, stained by Mallory's connective tissue stain, is repeatedly seen in amounts greater than in the bladder-wall of youth. I am inclined, therefore, to believe that although the senile bladder having no obstruction may undergo atrophy to a degree similar to that of the whole muscular system, at the same time there is apparently a tendency to a true bladder sclerosis, an irregular growth of new connective

tissue within muscle bundles, and at the expense of the fibres. This could be explained on the ground of probability as being a sequela of chronic inflammation, but that repeated sections show this quantitative change without submucous or subserous fibrous thickening or any remains of round-cell infiltration.

III. The presence of sclerosis in the vessels and walls of the senile bladder: Much complicated and ingenious theorizing has been devised to demonstrate a sequent connection between such a sclerosis and the conditions found in prostatism. For example: A senile change of both bladder and prostate; more specifically, changes in structure in the parts in question which depend upon a general arterio-sclerosis with modified blood-supply, — that is, that prostatism depends upon the local effects of a remote or general cause. A modified form of this theory declares that prostatism is a result of sclerosis of the vessels of the uro-genital system only.

Another subdivision under this theory makes the process a local one consisting in so-called "sclerosis" of bladder and parts connected, that is, an increase of connective tissue in the bladder-wall, at the expense of the muscle, with contraction in places, lack of tone and distention in other parts such as the formation of a pouch behind the prostate and the constant pressure of residual urine and its consequences.

I find that cases with intima and media thickened, and lumen correspondingly small, show the fibrous change in the muscles. On the other hand, cases with little or no change of vessels within the bladder-wall may show a considerable loss of muscle-fibres and corresponding new interfibrillary or interfascicular connective tissue.

It seems apparent then that there occurs in many senile bladders a quantitative change in the structure of the walls not due to inflammation, and that this change, while it may correspond remotely to the general muscle-changes of old age, does not depend on arterio-sclerosis within the bladder wall.

IV. As to the role of urethral obstruction in the senile bladder: In the development of prostatism and of the vesical

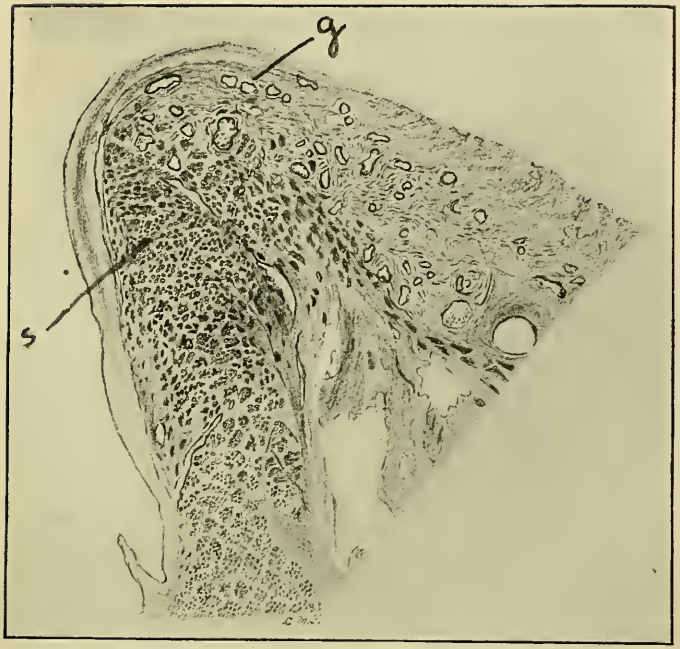

FIG. 3. Showing the posterior orificial lip. At the left, forming the anterior wall of the retroprostatic pouch, is the wedge-shaped section of sphincter (*s*). Superiorly, just under the urethral mucosa, are the few isolated prostatic glands (*g*), which are always present, which serve as origin of the third lobe.

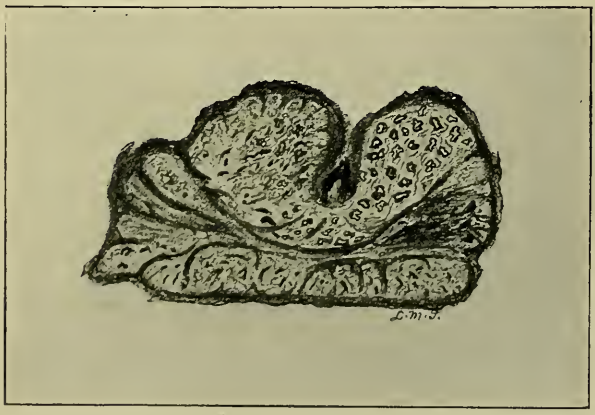

FIG. 4. Showing a fairly distinct horizontal transverse fibrous partition in a coronal section

pouch we have seen that the first element, and the one which may alone be sufficient, is atrophy of the bladder-muscle and replacement of the muscle by fibrous tissue. Such a bladder is less elastic than the unimpaired bladder; it tends more frequently to empty itself, and contracture, the usual role of new connective tissue, takes place. Grossly, fibrous bands intersect and form sharp, submucous ridges (Fig. 1). This more or less inelastic sac now no longer tends to maintain its natural ovoid form, and, weighted with urine by force of posture and gravity, it falls toward the rectum and perinæum. The orifice of the bladder, however, the beginning of the prostatic urethra, situated above the isthmus of the prostate at the apex of the trigonum, is fixed. The prostate is held firmly in relation to the pubes by the pubo-prostatic or anterior ligaments of the bladder. At this point, slightly posterior to the isthmus, in the plane of the posterior urethral sphincter, there forms an orificial lip, behind which falls away the pouch. To form this lip there need be no prostatic enlargement; in fact, the total volume of the prostate may be diminished. On section sagitally (Fig. 3) this lip is covered with mucosa, and immediately beneath it, and extending forward to be continuous with the prostate, is always a collection of prostatic glands. They may be few in number, at times dilated, but always present. Beneath them, and cut transversely, is the more or less wedge-shaped section of the inferior part of the posterior sphincter (Fig. 3–s). From this condition all stages up to the development of a true middle lobe may be seen. And this form of enlargement, like other forms of clinically enlarged prostate, is essentially glandular. Ciechanowski has shown successive stages of the development of a nodular middle lobe in this situation by dilatation of this small group of glands (Fig. 3–g) just within the posterior half of the sphincter, and by increase of their surrounding tissue, extending downwards and pressing always on the sphincter till the latter is fully and permanently dilated, is atrophied, and finally, under this middle lobe, disappears. This prevents effective shutting off, and the prostatic urethra is likely to remain filled with urine and constant desire to micturate be present.

This nodular form of prostatic outgrowth may appear laterally and so deviate or distort the urethra. If there is a real obstruction to outflow, and not mere deviation of the urethra, and there be any reactionary power in the bladder wall, its muscles will hypertrophy. Age and inflammation (*vide V., infra*) will limit or destroy this power to hypertrophy. And, furthermore, hypertrophy of bladder-muscles already existing may yield rapidly before foul and persistent cystitis. Cases which show merely the posterior orificial lip and pouch behind, without nodular or valve-like urethral obstruction, never show bladder hypertrophy.

V. On the role of chronic inflammation in the senile bladder: Chronic cystitis is an important secondary cause of vesical insufficiency. The usual remote origin of the cystitis is a persistent and sometimes latent gonorrhœal infection; a continual aggravating cause is catheterism. As has been already stated, there is no evidence, gross or microscopic, that such inflammation is an essential in the changes observed in insufficient bladders. The bladder-wall in cases of long standing cystitis may show the greatest degree of thickening. This thickening on section is made up partly of a round-cell infiltration of mucosa and submucosa, but mostly of a considerable submucous development of fibrous tissue, which in old cases raises all over the inner aspect of the bladder (Fig. 1) a tangle of crossing ridges. These ridges may be so prominent as to convert the inner aspect of the bladder into a succession of pockets or saccules. There is some new connective tissue formed between the bundles of muscles, with round cells here and there through it, which show its inflammatory origin. New subserous fibrous tissue may less commonly form part of this thick wall.

The importance of chronic inflammation in the senile bladder lies principally in the fact that it rapidly intensifies all the changes and symptoms already existing. The bladder suffering from it is less tolerant, contracture is greater, atrophy is more rapid.

VI. On the presence of stone in the senile bladder: This is of great frequency but often undemonstrated before operation, because (*a*) the symptoms of stone are masked by

those of obstruction and cystitis; (*b*) the calculus forms and remains in the retro-prostatic pouch and may be even quite enclosed in one of the saccules referred to. Large stones felt by rectum sometimes are mistaken for enlarged prostate.

CONCLUSIONS.

(*a.*) Senile vesical insufficiency, which may be called clinically "prostatism," is not a single entity, but a complex condition. It has a distinct anatomical basis, and may be due to one or more of several causes.

(*b.*) It is not due to fatty degeneration or infiltration of bladder-muscle.

(*c.*) It is not due to arterio-sclerosis of bladder-vessels. Changes in walls of blood-vessels have no constant relation to change in the bladder-wall.

(*d.*) The causes of bladder insufficiency are:

1. Atrophy of bladder muscle and new growth of connective tissue, finally infiltrating the muscles.

2. Mechanical obstruction at the beginning of the urethra. This may be simply a posterior lip at the orifice in front of a retroprostatic pouch; it may be a true middle lobe of the prostate acting as a valve; it may be a nodule or enlargement of one or both lateral lobes protruding into the urethra.

3. Chronic cystitis intensifies these two causes, namely, muscle atrophy and obstruction at the exit, by means of the structural changes due to inflammation.

4. Stone in the bladder increases the symptoms, but is probably rarely a primary cause of insufficiency.

(*e.*) Although structural changes in the bladder wall may alone be sufficient to cause prostatism, the most important factor is probably obstruction.

(*f.*) The tendency of all these changes increases with age.

And, finally (*g*), the most marked cases are those where atrophy of bladder muscles, connective tissue infiltration, enlarged prostate and chronic inflammation are all present.

Part II.

ON THE STRUCTURE, GROSS AND FINE, OF ENLARGED PROSTATE.

A. *The Normal Prostate.* — The normal prostate is classically and well described as a chestnut-shaped body adjoining the neck of the bladder, inclosing the first part of the urethra. The urethra runs nearer the anterior than the posterior aspect of the gland, that is, about .7 cm. from the former and .9 cm. from the latter, but it varies (25). The narrow end or apex of the prostate is directed downwards and forwards to within 1.5 cm. of the symphysis, to which it is connected by thickenings of pelvic fascia. The posterior part or base is close to the rectum, through which it may be felt, about 6 cm. above the anus (48). Its measurements are about 3.5 cm. across at its widest, *i.e*, bilaterally; 3 cm. from base to apex, and about 2.5 cm. in thickness, *i.e.*, in the direction of the urethra. The weight of the normal prostate ranges from 13.6 grams to 21.4 grams, the average being 18 grams (72). These limits are not strict. A sheath derived from the recto-vesical fascia encloses the prostate and contains the prostatic veins. This capsule is fairly distinct. The rectal surface shows two grooves which meet in front and indicate the course of the seminal ducts. The gland presents a lateral lobe on each side of the base, and a middle portion or isthmus which is included between the ejaculatory ducts and the neck of the bladder.

The prostatic urethra, about 3 cm. long, is dilated in its middle, where there arises from its floor the verumontanum (colliculus seminalis, caput gallinaceum) (69). The ejaculatory seminal ducts, passing forwards from the seminal vesicles, traverse the lower part of the prostate and empty into the urethra by two slit-like openings very near the crest of the caput. The prostatic ducts empty through the wall of this portion of the urethra by twelve to twenty (15 to 32, Svetlin, quoted by 75) openings, most of them on the floor of the urethra in a fossa on each side of the caput, called the prostatic sinus. The two largest prostatic ducts

empty at the caput just behind the mouths of the ejaculatory ducts (Walker, loc. cit. 253). In the middle line, close in front of the orifices of the ejaculatory ducts, in the forward part of the caput, is the prostatic utricle (sinus pocularis, uterus masculinus). This is a blind pocket, about .8 cm. in length, extending upwards and backwards beneath the isthmus.

Histologically speaking, the prostate normally is made up of smooth muscle fibres, of rather cellular connective tissue, and of true gland-tissue in the proportions roughly of 1:1:2, together with more or less elastic tissue. On examination of a section here and there, no idea of a definite order of arrangement of these elements may be obtained. Walker (loc. cit. 244) by a most careful study of serial sections in the dog, has come to the following conclusions with regard to the distribution of

Muscle in the Prostate. — "That the vesical end of the prostate is surrounded by a thick muscle composed of longitudinal, circular and oblique fibres, from which a large process projects anteriorly and posteriorly between the two glandular hemispheres; and a thick sheath is sent out on either side, which encases the fore, lateral and dorsal surfaces. The posterior process extends to the urethral end, where it spreads out round the external surface in that region. From these muscular divisions, septa pass into the gland and surround the lobes in a circular and longitudinal manner, giving to each lobule two distinct coats. The muscle-coats of the urethra and bladder are inserted into the prostate and not continued through it, and the urethral coats in the prostatic portion are replaced to a considerable degree by prostatic substance." Striated muscle in the prostate is found only as a part of the external sphincter of the bladder at the urethral end of the gland, principally as a thick layer of circular fibres.

The Connective Tissue part of the stroma can be only fully recognized and duly appreciated when stained by Mallory's connective tissue stain. It may then be seen to form one-fourth or less of the gland, a fairly dense, fibrous structure, rich in cells at birth and in youth, becoming more fibrous as age advances. On gross coronal section (Fig. 4) in the

normal prostate of middle life and later, a distinct transverse, horizontal, fibrous partition is seen. This runs in a plane across the gland about one-third of the distance from the urethra to the posterior surface, and as it approaches the sides, inclines downwards toward the latero-posterior angles. Branches of this plane often curl upward to more or less encapsulate the lateral lobes, merging finally with the sheath.

Microscopically, these septa, more dense and fibrous than the rest of the connective tissue stroma, send out in all directions ramifications of increasing fineness around and between the muscle-masses, between and supporting the gland lobules. This tissue accompanies the innumerable capillaries of the organ. Between these smallest vessels and the cellular stroma in dogs, Walker finds a membrana propria made of finest connective fibrillae. Regnauld (67) quoted by Walker, finds none; Langerhans cannot differentiate it; Walker cannot demonstrate it in the human. A few small, round cells, plasma cells and leucocytes are to be seen normally, but in the larger septa, rather than near the lobules, a point of difference to be noteworthy later.

Elastic Tissue, in the normal gland, has been carefully observed by Walker (loc. cit. 248), who also quotes Antonini (5). Walker finds surrounding the urethra, just under the mucosa, a sheath of longitudinal fibres. The outer fibres spread into the gland and form a figure-of-8 network round each prostatic duct, apparently making an elastic sphincter for each. Extensions are seen into the larger fibrous septa, and from these fine fibres arrange themselves in a circular manner round the alveoli (Fig. 5). The caput is especially rich in elastic fibres. They are arranged round the utricle about each ejaculatory duct, besides a considerable submucous layer of it all over the prominence.

The Glandular Portion of the normal prostate consists of thirty to fifty simple tubes, straight in the embryo, later branched, and finally forming complex lobules. They unite into a smaller number of excretory ducts, and finally empty into the prostatic sinuses as already described. In the upper portion of the prostate the alveoli are smaller and more saccular, smallest at the exit. In the lower part of the gland

Fig. 5. Showing the distribution of elastic-fibres in the normal prostate.

Fig. 6. Showing the senile caput with its apical duct, amylaceous bodies, and a slight degree of small round-cell infiltration.

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the tubules are longer (71 and Quain, loc. cit.). In the anterior commissure the tubules are relatively few in number, and simple.

The epithelium lining the glands in the dog have been described with the greatest exactness and detail by Walker, and in man by Langerhans (42). The cells are arranged in one layer, and vary in shape — long columnar, cuboidal, or even irregular, with relatively large nuclei near the base in the primary lobules, while in the ducts the cells are more flattened with nuclei near the centre, and at the exits are merely squamous, of the urethral type. The alveolar secretion is seen as a shrunken, finely granular mass, staining pink by eosin. An occasional desquamated granular epithelial cell is seen, and in senile prostates, which in no other way deviate from the normal, there are so frequently seen round, stratified, so-called amylaceous bodies, that they must be mentioned in describing the normal prostate. They vary in size up to filling of the saccule, and consist often of clearly defined concentric strata. Some stain red, others blue by Mallory's connective tissue stain.

The *caput gallinaceum* is made up of connective tissue and muscle, together with a relatively large amount of elastic tissue. The glands of it are near the summit, and open by a common duct just anterior to the centre of the crest (Fig. 6). Muscle surrounds these glands as elsewhere, but does not extend to near their orifices.

Adenoid tissue described by Walker (loc. cit., p. 250) I have not seen. He says: "In quite a number of prostates, bits of adenoid tissue were scattered here and there throughout the tissue, generally near the lateral surfaces. Two or three small nodes are usually near together, with a rather thick layer of connective tissue between them. In some of the nodes are minute channels, lined by endothelium; these are most probably lymph vessels."

The *vessels* of the prostate are branches of the vesical, hemorrhoidal and pudic arteries. They pass into the prostate along with the larger connective tissue septa, where they break into smaller twigs, follow the ducts to the lobules and break up into capillaries about the alveoli (64). On the

sides and base of the prostate, in its fibrous sheath, the veins form a plexus. This is highly developed in old subjects. These veins behind pour into branches of the internal iliac. In front they empty into the dorsal vein of the penis.

Lymph channels and vessels may be found between the two layers of the fibrous sheath.

The *nerves* are derived from the hypogastric plexus of both sympathetic and central origin. They contain both medullated and non-medullated fibres, and show here and there ganglion cells. They are seen in the posterior and lateral surfaces and pass along the fibrous trabeculæ towards the alveoli. Their course is extremely difficult to follow, and practically nothing is known of their termination.

B. *The Senile Enlarged Prostate.* — The character of the changes which together may be seen to make up the structure, gross and fine, of the enlarged prostate, is to be most clearly understood by a separate presentation of the changes observed in each structure, particularly the glands, the muscle and the connective tissue.

Gross. — The senile prostate which has suffered general enlargement attains a greater size than that in which one lobe has increased in size. The limits of *weight* of Thompson's cases were 22 to 180 grammes; of mine, 27 to 180 grammes.

The *capsule* varies much in strength and thickness — in some cases the gland may be easily shelled out, in others it is hard to differentiate gland from capsule.

The *consistence* of the enlarged gland may be extremely spongy, through degrees of increasing firmness, up to a hard, fibrous feel. The gross appearance of a *cut section* corresponds to these differences in feel, that is, from that of a fine sponge to that of a cross cut of a fibroid of the uterus. Some show on the cut surface many distinct, encapsulated, round, fibrous bodies, which protrude above the level, as if relieved from the compression of a tight capsule. This is the "Knotige" form of enlargement described by the Germans. From all, except the hardest, there comes from a cut surface on squeezing a slightly gelatinous turbid fluid.

The *distribution of the enlargement* is irregular. It appears most often in the lateral lobes, next often in the

lateral lobes and in the form of a middle lobe, next often as a middle lobe alone, and lastly as one lateral lobe. The anterior commissure rarely suffers enlargement, a fact of considerable significance, as will be shown.

It is now necessary briefly to consider the gross details and immediate mechanical effects of enlargement of the prostate in each of the varieties just mentioned.

1. *Bilateral Enlargement.* — In this form the urethra is increased in length, and its cross-section is transformed from a triangle to a vertical slit. To do this, the side-lobes have grown downwards and backwards, and the floor of the prostatic urethra is depressed below the level of the posterior lip of the bladder orifice. In this manner the posterior obstructive lip (Mercier's bar) can form even when the hypertrophy is of the lateral lobes; the sphincter remains normal, while the urethral floor anterior to it is depressed, and a bar is formed without the necessity of bringing in an hypertrophied sphincter to explain it. It is in this way, together with the bladder wall changes, that retention begins in cases of side-lobe enlargement.

2. *The Formation of a Middle Lobe.* — This takes place either as a part of a general enlargement or alone. Its interest grossly is in its relation to the posterior half of the sphincter muscle at the orifice. The older observers (16) held that the middle lobe arises from the portio intermidia, anterior to the sphincter, and grows up between the urethral mucosa and the posterior half of the sphincter. The sphincter thus comes to lie always behind a sort of valve-like tumor, later becomes flattened out, and finally disappears through atrophy. More careful later observation (40) makes it probable that this polypoid middle lobe has nothing to do with the posterior commissure or isthmus, which is always below the sphincter, but that it develops from the few isolated, prostatic acini which lie between the mucosa and the sphincter at the orificial lip of the the bladder (Fig. 3g). All agree that the sphincter posteriorly diappears, either being pressed upon by the new growth above it, or suffering infiltration by the growth. This middle lobe may act as a cause, therefore, of either retention or incontinence; retention

if the growth fit like a ball-valve exactly into the orifice, — incontinence, if it does not fit the orifice and at the same time does continuously stretch and so destroy the tonus of the sphincter. If besides the pressure from above, from the middle lobe, there is also pressure from below and in front from enlargement of the rest of the prostate, the sphincter becomes pushed up into the true bladder-wall. Thus, so far as function goes, the sphincter disappears because in this position it has no relation to the orifice and the bladder must be then continuously incontinent unless there be a valve. At this point, however, it must be made finally clear that these ball-valve growths which arise from the relatively isolated prostatic glands under the mucosa of the orifice, are to be distinguished from the point of view of mechanics, from the posterior lip already described. This barriere vésicale of Mercier is a mechanical obstacle; it favors the formation of a pouch, but its pathogenesis is that of the third lobe. They both arise from dilatation of prostatic glands, those of the valve-like lobe coming directly from the isthmus and tending to fill the exit, while the posterior lip obstruction is somewhat behind the orifice. It is definitely clear that this bar is never due to hypertrophied sphincter.

3. *Enlargement of One Side Lobe.* — This is relatively uncommon. It develops in the direction of least resistance, which seems at first to be toward the periphery, but finally is always towards the urethra where it narrows that passage, lengthens it and causes it to deviate laterally, according to the contour of the new growth. Unless it be sufficient in amount actually to interfere with the patency of the urethra, there is likely to be little retention or incontinence, unless there be also some of the other causes present in the sphincter or the bladder-wall.

4. *General Enlargement.* — This is one of the common forms where, besides bilateral enlargement, there is also developed a third lobe. The bladder-pouch appears early and the urethra suffers narrowing from side-pressure, or is obstructed, or is held patent according to the final relation in size between the enlarged portions of the prostate in a given case.

FIG. 7. Showing areas of scar-tissue, round-cell infiltration, and glands wholly or partly denuded of ephitelium.

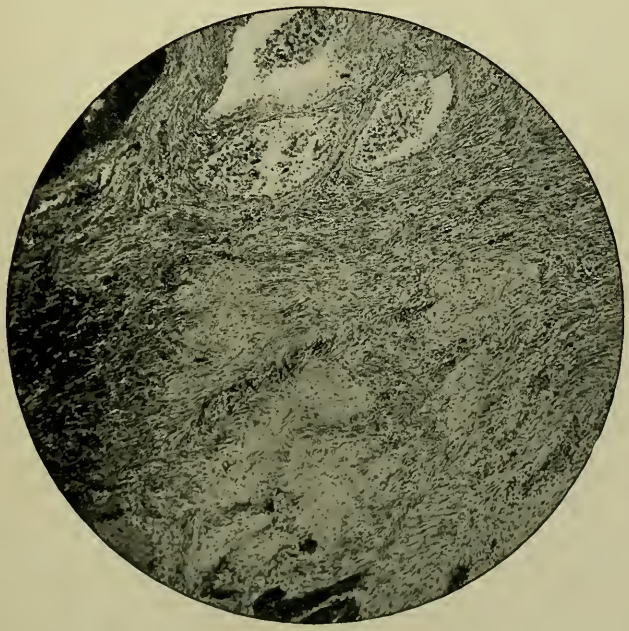

FIG. 8. Showing well-marked scar-tissue areas, and denuded glands with pathological contents.

In short, the changes in function due to general enlargement are a combination of the changes seen in enlargement of the separate parts.

Microscopic. — It now remains to go into a detailed microscopical study of the enlarged prostate.

Proportion of Constituents. — In the normal prostate, as we have seen, the proportion of muscle, connective tissue and gland tissue is roughly 1: 1: 2. In the enlarged prostate, in practically all cases, the observer is at once impressed with the relative increase in the area taken up by glands; no definite proportion, however, can be worked out (55).

Muscle in Enlarged Prostate. — The muscle-sheaths which start at the vesical end remain about fixed in size but often show some fibrous infiltration. The coats which surround the acini and ducts no longer show a clear division into two coats, in fact, more often round dilated glands, only indistinct traces of muscle tissue remain. Remains of the larger septa from which the coats that surround the glands arise may be seen cross cut here and there in nearly all sections. The cells stain well, nuclei show no active subdivision and a close intermixture of fibrous tissue with each bundle exists. Many groups of elongated, spindle-shaped cells, which I believe hitherto to have been considered new smooth muscle cells, stain blue by Mallory's connective tissue stain and they should without any doubt, therefore, be classed as new connective tissue. About some glands may be seen portions of muscular capsules occasionally thicker than those in the normal gland. This is the only suggestion of true "hypertrophy" to be seen in enlarged prostate.

The Connective Tissue, part of the stroma, may be greatly increased in relative quantity. The average amount could be roughly estimated as two or more times as great as that of muscle. It is richly cellular for the most part, but presents two differences from that of the normal prostate. The first of these differences, the real importance of which was first made clear by Ciechanowski, is the presence of small round cells (Fig. 7). These cells are seen most often in the fibrous tissue near the acini; they may be diffusely distributed (Fig. 16), and isolated groups of them are also

seen. Such groups may, of course, be in relation to glands not cut by the section. The groups are entirely irregular in outline and present no suggestion in the way of minute channels or pellicle, of the lymph-nodes of Walker. The nuclei are single, relatively large, stain deeply with basic stains, and the cells cannot be distinguished from lymphocytes. They are similar in kind and distribution to those seen in chronic mastitis. Examination of every large prostate shows these cells. Many cases show undoubted, polynuclear, round cells.

The second distinguishing feature of the new fibrous tissue is the presence in it, here and there, of irregular masses of nearly hyaline, nearly homogeneous, dense, poorly staining fibres, in short, masses of scar-tissue (Figs. 7 and 8). About such scars small round cells may be seen. The masses may be in relation to tubules or apparently apart from them. In the midst of such masses of scar-tissue may nearly always be found remains of glands (Fig. 8). Such gland remains are usually denuded of epithelium or collapsed by pressure, or, in cases, the presence of a so-called amylaceous body may alone show the previous presence of a gland. Within the scar-tissue may also be seen clumps of the spindle cells of new connective tissue or small areas of round cells.

Such associated appearances, namely, small round cells, well staining spindle cells and scar-tissue can be looked upon probably as stages of one inflammatory process.

The relation of these stroma changes to the glands is the next question of importance. In general, all the changes named are seen to a far greater extent near the central or urethral part of the prostate than near the periphery. The round-cell clumps in particular are seen repeatedly near the walls of the larger terminal ducts (Fig. 9). In addition there are often areas infiltrated with polynuclear leucocytes. These areas are of any size, even up to small abscesses visible to the naked eye (Fig. 10). Fairly definite rings of the new connective tissue cells are seen in places near ducts and the scar-tissue, as has already been noted, may be found many times quite enclosing and compressing a duct of mod-

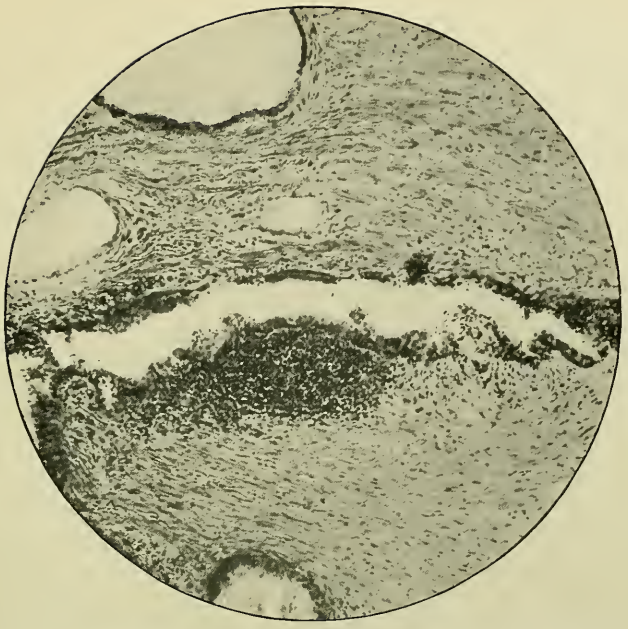

FIG. 9. Showing round-cell infiltration with minute abscess formation in the wall of a prostatic duct. Dilated and denuded glands nearby.

FIG. 10. Showing microscopic abscess formation surrounding and including a prostatic gland.

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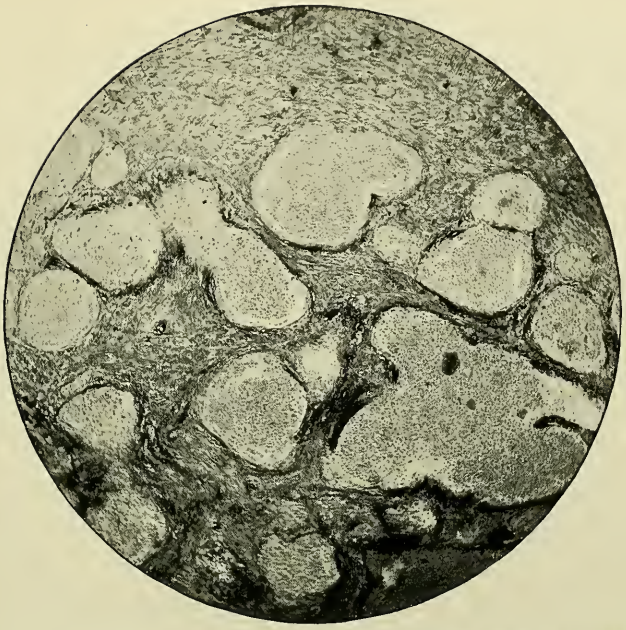

FIG. 11. Showing bundles of elastic tissue squeezed between the dilated glands of enlarged prostate.

FIG. 12. Showing a stage in gland-dilitation; remains of gland-partitions in the form of shelf-like projections; in one place a single compressed line of connective-tissue supports epithelium on each side of it.

erate or large calibre, some of these even to complete obliteration. These evidences of proliferation in the stroma are seen, as a rule, not between or round the glands which have suffered dilatation. This is a point of great importance, as will be seen in considering the changes in the glands in an enlarged prostate.

Elastic Tissue in the Enlarged Prostate shows little or no change in quantity (53) but stains less sharply near dilated glands or near those which have purulent contents. The elastic fibres (Fig. 11) are less wavy and are more closely compact in bundles as if pressed upon laterally by the dilated glands on one side and the stroma on the other. They form, therefore, in the enlarged prostate, narrow dark bundles following the outline of the enlarged acini.

The Glands or Acini in Enlarged Prostate. — As has been said, the first impression on examining sections of most enlarged prostates is the apparent relative increase in the area covered by glands. The ducts are in many places wider and the glands are much dilated (Fig. 13). Two glands close together dilate till they are separated only by their respective linings of epithelium, and one or two degenerated supporting connective-tissue fibres between them (Fig. 12) ready to give way. In others, a shelf of fibrous tissue protruding into a lumen is the only remains of a previously existing partition. Other parts of these acini show changes due to dilatation also. The *epithelium* lining the glands and ducts of the enlarged prostate presents always marked changes. The cells are smaller, they are flattened, their nuclei stain less deeply, the cytoplasm becomes granular and stains faintly. In places (Figs. 7, 12, 13, 14) the cells have flattened until they are scale-like; in places they have desquamated. In some glands a series of six or more cells still connected together in a row may be seen lying free in the space near the place in the wall from which they have been separated. In other glands, a few flattened nuclei in the wall are the sole remains of the lining membrane, while the final stage of these changes shows no epithelium remaining, but only the gland contents lying free in a space in the stroma. In some of the acini, the

degenerated lining-cells desquamate and coalesce in an ill-defined, poorly-staining, granular or even amorphous mass in the centre of the dilated space, or in other glands whether or not the wall has been denuded of epithelium, there are seen the so-called "amyloid bodies" or concretions. These are more or less stratified rounded bodies, staining red or blue by Mallory's connective tissue stain, and contain within themselves small, granular bits of chromatin, often enough to suggest their origin in old epithelial cells. These bodies are constantly found in dilated glands of enlarged and normal-sized prostates, so often indeed as to warrant the belief that they are really a normal senile condition.

The granular or amorphous mass in the dilated gland-space may also show small, round mono- or poly-nuclear cells (leucocytes) in varying numbers, even up to an amount to make the glandular contents truly purulent. This is particularly seen in the larger spaces. The larger gland-spaces always have pathological contents, that is, epithelial products together with few or many leucocytes; they are never empty. The size of these dilated glands varies much, from the slightly distended tubule up to a microscopic cyst or abscess. The form of the glands remains normal with some distention, but as the internal pressure increases they become quiet round on section in every plane.

It is apparent, therefore, that the view held by Socin (69) and Ciechanowski is amply supported. In enlarged prostate, the essential change is a passive dilation of the prostatic glands and ducts induced by the gradual accumulation in them of retained secretion, degenerated cast-off epithelium and leucocytes. The process is probably not a physiological one, because the greater the degree of dilatation the gland has suffered, the greater, as a rule, the number of leucocytes in it. The amount of cast-off epithelium in the gland-spaces suggests an increased activity of production of epithelium, it is true, but not neoplasmic in character. The lining membrane in their sections is only one cell thick, and the proliferation arises from cells which do not show the staining reactions of vigorous epithelium. It is as if an increased rapidity of production of cells had taken the place of production of the usual secretion.

Fig. 13. Showing the single row of flattened epithelium in a dilated gland; glands in various stages of dilatation; round-cell infiltration.

Fig. 14. Showing glands dilated, with flattened epithelium; other glands and ducts compressed by fibrous tissue and showing subepithelial round-cell infiltration.

The distribution of the glandular dilatation in enlarged prostate is a matter of the utmost importance and significance. Under this heading four observations are to be separated:

1. Many enlarged prostates, on gross section, show here and there all over the cut surface, small but still macroscopic, round, fleshy nodules or kernels looking like minute fibroids. Microscopically, these always have for their essential structure one system of enlarged acini. These are the "pseudo-adenomata" of Ciechanowski. Each consists apparently of all the elementary glands which originally arborated into a single duct; obstruction of this common duct by contraction of the new fibrous tissue about it has caused retention of the secretion of all the tributary acini, and their passive dilatation has resulted. In the dilatation the muscular and elastic layers surrounding each gland of this individual system of glands have been stretched out, have lost their infoldings, and remain finally as a distinct spherical capsule round a mass of dilated glands.

Such "pseudo-adenomata" may be seen well distributed all through the two side-lobes and may be the essential factor of their general enlargement. One lateral lobe alone may suffer enlargement of this type, and much less commonly a middle lobe may develop in this manner.

2. Another principal, but never distinct, form of enlargement is where the obstructive dilatation of glands does not take place in isolated minute gland-systems, but shows itself in glands here and there not drained by a common duct. The picture then shows, in one place, one or more relatively large cysts or abscesses; in another place, glands squeezed to partial or complete obliteration (Fig. 14); in another place a perfectly normal area.

In describing these two forms of microscopic change, that resulting in the formation of knots or nodules, and that which is more diffuse and irregular, not obstructing definite gland-systems, it is quite apparent that the difference is not one in the kind of change, but in its distribution. There seems to be no pure form of enlargement. Every case presents a mixed form, and the different

varieties may be seen in one section. It should be here observed that there seems to be no sound evidence that the obstruction to free drainage of prostatic acini lies anywhere but in the surrounding stroma. It has been suggested that the amylaceous bodies obstruct the ducts, but this cannot be true in most cases, because the presence of these bodies is inconstant within the dilated glands, and, furthermore, in half the glands the bodies are seen in a proximal or peripheral position where they could not obstruct. Analogous examples of obstruction of exit-ducts by pressure of contracting stroma are seen in the formation of minute cysts in chronic mastitis and in varieties of chronic nephritis.

3. For prostatic enlargement to involve the anterior commissure is exceedingly rare. As has already been suggested, this fact seems particularly significant. Ciechanowski (1, p. 266) has collected only seven cases in all the literature where the anterior commissure was affected, and he quotes Aschoff (8), who has shown that only a few or no gland tubules exist in this portion of the prostate. If, now, these glands or their muscular coats caused prostatic enlargement by neoplasmic growth, there is no reason why such development, that is, a multiplication of glands, should not take place in the anterior commissure as well as in other parts, but inasmuch as passive dilatation of already existing glands is the cause of enlargement, these glands of the anterior bridge, the same in origin and structure as all other prostatic glands, are merely too few in number and too small to be able to exhibit macroscopic changes. There are rare exceptions to this rule.

4. For the last and most important observation on the fine distribution of the proliferative changes within the senile prostate, all credit for originality is, without question, due to Ciechanowski. In enlarged prostate, the new connective tissue, the contracted scar-tissue and other signs of proliferation are seen most abundantly in the deepest portions of the gland, that is, midway between the periphery and urethra, extending usually towards the urethra. There is, then, obstruction of exits, that is, of distal ducts, those nearest the urethra, and consequent retention of glandular secretion and

other products of epithelial activity in the proximal or peripheral parts of the gland-system. The obstruction is gradual, and, at this period, the muscular coat of each gland may hypertrophy to expel the glandular contents against opposition. Complete obstruction being established, passive glandular dilatation proceeds and general enlargement results.

When, now, atrophic prostates, that is, those below normal in weight, are studied (1 p. 282), unexpected and ample confirmation of this simple hypothesis of an almost mechanical method of production of prostatic enlargement is found.

Abnormally small prostates seem to be of two varieties. The first includes those which accompany lack of development or loss of the testes. In this variety, the tubules are narrow, are only elemental in branching, and the epithelium is small and not active. The second variety of small prostate is found in old men where the testes are normal, and on section such prostates appear in many respects similar to the enlarged ones. The small round-cells, the new connective tissue, the scar-tissue and the amylaceous bodies are all present. The glands, on the other hand, are small, compressed. The new contractile tissue has formed round the primary acini, in other words, more in the periphery of the prostate. The glands at their origins are compressed, their secretion, far from being retained, is expressed, and the whole prostate diminishes in size.

It seems warrantable, with regard to *Conclusions on the Histogenesis of Enlarged Prostate*, to add such weight as I may, as have Greene and Brooks (27) recently, by reaffirming the conclusions of Ciechanowski:

The so-called enlargement of the prostate, as well as certain forms of prostatic atrophy, are related histogenetically, and have a common cause. The two processes do not differ qualitatively but only in the distribution, intensity, and localization of otherwise analogous changes.

The common starting point of the enlargement and certain forms of atrophy is to be sought in the productive connective tissue processes which occur in the stroma, and according to the stage of their development can show different stages of

repair, but are always in isolated masses, and principally arise directly under gland epithelium.

If the stroma changes are located in the central prostate near the principal exits, the lumina are closed, secretion collects and peripheral dilatation results. This dilatation is the more rapid and reaches a higher degree, the more numerous and the nearer to the exits of the principal tubules the obstruction occurs, and also the higher the degree of the simultaneous intraglandular pathological processes. These consist mostly of active proliferation, followed by desquamation called catarrhal, and may end in a purulent process by the accession of leucocytes.

The enlargement of the prostate is almost exclusively due to dilatation of glands. The new-formed connective tissue is relatively unimportant, and the active participation of muscle tissue in enlarged prostate in the way of true myoma (great majority of cases) is unproved and doubtful.

If the stroma changes take place principally in the periphery and near the blind ends of the tubules, then there is atrophy of the tubules, shrinking of the connective tissue in the stroma and atrophy of the entire prostate. This is quicker and more intense if there is no endoglandular pathological process.

According to the balance of these two processes the prostate may be of normal weight, increased or diminished.

Atrophy and enlargement are histogenetically and probably etiologically similar.

Part III.

ON THE CAUSE OF ENLARGED PROSTATE.

Since the earliest times the distressing symptoms of enlarged prostate have been observed and their cause sought. According to Bernays (8), the earliest mention of the prostate as the cause of many of the senile bladder symptoms was in the writings of Morgagni, printed in 1761. His opinion was corroborated by John Hunter in 1786, in his "Treatise on the Venereal."

It seems necessary, in considering the etiology of enlarged

prostate, to take the space here first to summarize briefly the beliefs of the best observers on this subject, because even now widely divergent opinions are still held.

One of the earliest opinions seems at the present time, as is not infrequently the case, to be one of the best. Desault (15), a follower of John Hunter, in 1813, first suggested the inflammatory origin of enlargement of the prostate, saying that it was common in those who have had many attacks of gonorrhea.

Home (38), at about the same time, proposed as the underlying cause, the habitual congestion of the parts about the bladder-neck, such as might be brought about in high livers or in people who sit much.

Mercier (54), in 1841, considered the cause to be sluggish circulation of the parts, such as one expects in men with much fat, or with sedentary habits. Such men, he says, have relaxed veins and most often suffer from prostatic enlargement.

Astley Cooper (13), in 1824, declared the affection was the result of age alone and only physiological. In this view, many have concurred, particularly French writers. Thus Launois (43), Regnauld (67), and Guyon (32), have all contended in recent years that prostatic enlargement is a senile involution in the physiological sense.

Next, there are many (4, 7, 19, 21, 26, 28, 36, 39, 44, 45, 49, 57, 58, 62, 77, 78, 79) who base their theories of prostatic enlargement, in one way or another, on the apparent parallellism of development and function between the prostate and the testes. Lydston (46) combines congestion with sexual "prostatic overstrain" as a cause.

Syphilis, gout, rheumatism, stone and stricture have been considered amongst etiological factors, but are spoken of now only to be at once excluded since the beginning of modern pathological histology.

The bearing of arterio-sclerosis, local and general, and of bladder-wall changes, we have already considered at great length, and have decided that, as a constant etiological factor in the production of actual increase in the size of the prostate, it may be ruled out. It is seen in the enlarged prostate (Fig. 16) and in that of normal size.

Finally, there is the belief that the enlarged prostate is suffering from a true new growth, — single or multiple fibroma or fibromyoma. Of the adherents of this theory, the most prominent is Thompson, who says (72, p. 131): "Inflammation must be eliminated from the category of alleged causes," arguing that chronic inflammation would cause eventual shrinkage rather than increase in size. He draws an analogy, already referred to, between the uterus and the prostate as to their origin, and states that, just as at the close of functional activity, the uterus is liable to develop new growths similar in structure to its own, so in the prostate, at a similar stage of development in the male, a similar proneness to growths like itself in structure may be seen. Recent writers who still believe in the fibro-myomatous prostate are few (35). Maguire probably best summarizes their view when he says (46), "Sometimes the obstruction is due wholly, or in part, to fibroma, single or multiple, of the prostate. They form distinct encapsulated growths. These tumors are like the fibro-myomata of the uterus; of their etiology I can offer you no conjecture."

As a last word in this list of hypotheses, it should be said that no one, so far as I can discover, still considers enlarged prostate to be a true hyperthrophy.

It only remains now to discuss briefly in the light of modern pathology some of the more important theories in the list above.

1. *The relation of the testes to enlarged prostate.* The theory of the dependence of enlarged prostate with its sequelae on the testes, is based upon (*a*) the parallel physiological development of the two; (*b*) on the arrest of development of the prostate after an arrest of testicular growth; (*c*) on atrophy of normal prostate after castration in animals and youths.

The observations on which this theory is founded are correct. There is undoubtedly in animals a near relation between castration or resection of the vas deferens and atrophy of the normal prostate. The histology of such prostates shows, as has been noted, a true atrophy of all elements. The supporters of the belief that prostatic enlargement, on

the one hand, is due to a senile diminution of testicular function, and at the same time, on the other hand, advocate castration as a therapeutic measure to retard the enlargement, contradict their own observation. For (1, p. 288) as a proof of the correlation between the development of the prostate and the testes, they bring forth the fact that castration causes prostatic atrophy as a result of loss of sexual function, while in the same argument they declare that prostatic enlargement is due to some senile loss due to diminished testicular activity. In short, they contend that the original prostatic enlargement or the later diminution after castration are both due to loss of sexual function. This position is not tenable. Moses (59) reports enlargement of the prostate in a man of sixty-eight some years after a double complete castration, thus proving beyond doubt that the changes seen in enlarged prostate undoubtedly develop independently of the presence or absence of the testes.

White (77, 78), Moullin (57) and Harrison (36) have been the most ardent champions of a therapeutic application of this theory, though I believe I am correct in saying that White no longer believes such an application to be uniformly indicated (79). White's belief was based on experiments upon dogs, and since his first reports several (12, 45, 67) have worked on similar lines. It appears to be now established that in all animals except dogs, castration is always followed by prostatic atrophy. In dogs the results are variable, one case for example (45), five months after double vasectomy, showed a very large prostate. It is to be noted at this point that the dog, which is the only animal where prostatic atrophy does not always follow castration, is, according to the veterinarians (1, p. 290), the only animal that suffers from prostatic inflammation.

Cabot (10, 11), too, entirely from the clinical point of view, has ably controverted the hypothesis of a connection between the testes and enlarged prostate. It is true that the prostate in the living, nothing being known of its histology, has, without question, after castration, diminished in size. But it is also true that enlarged prostate is much more often seen in the living than it is found on the autopsy table.

There is, in short, evidence that pressure and irritation and inflammation at the bladder-neck produce their usual sequelae of congestion, edema, and even evanescent hyperplasia, and that these conditions may all be relieved and made less by the diminution of blood supply and by the functional inactivity which must follow castration. Indeed, the diminution in size follows too quickly to be a true tissue atrophy; take, for example, a case operated by Gavin (25), where the patient, a few hours after castration, passed urine voluntarily for the first time in months. Cabot says "there are numerous cases in my practice in which removal of a stone had been followed by such subsidence of irritation at the vesical neck that no operation on the prostate was necessary," and he, too, concludes that "no more wholly unsupported theory has been advanced in the whole discussion than that quick relief which sometimes follows castration is due to a rapid process of atrophy."

Englisch in Vienna is now making a physiological clinical study of the relation between the prostate and testes, and Ciechanowski is also working on this problem.

2. *Is enlargement of the prostate neoplastic in origin?* Beginning with Velpeau (60) and Thompson, those who believe the enlarged prostate to be a true new growth, usually fibro-myomatous in character, are many, either by declaration or implication.

The facts that the prostate is purely a genital organ, as has been definitely established (75), and that it contains the uterus-masculinus, give ground for the reasoning that fibro-myomata may arise in the prostate analogous to those found in the uterus (35). Grossly, however, the largest prostate is only occasionally as large as the average uterine fibroid. Fibro-myoma of the uterus, even the intramural form, may in most cases be sharply differentiated, by the eye at least, as a definite tumor. The prostatic tumor, if a fibroid, must be considered atypically diffuse. Microscopically, the myomatous part of the uterine fibroid is more cellular and the nuclei are usually more uniformly distributed than in the prostatic tumor, that is, there are fewer of the hyaline fibrous areas resembling scar; and in the prostatic growth also, in

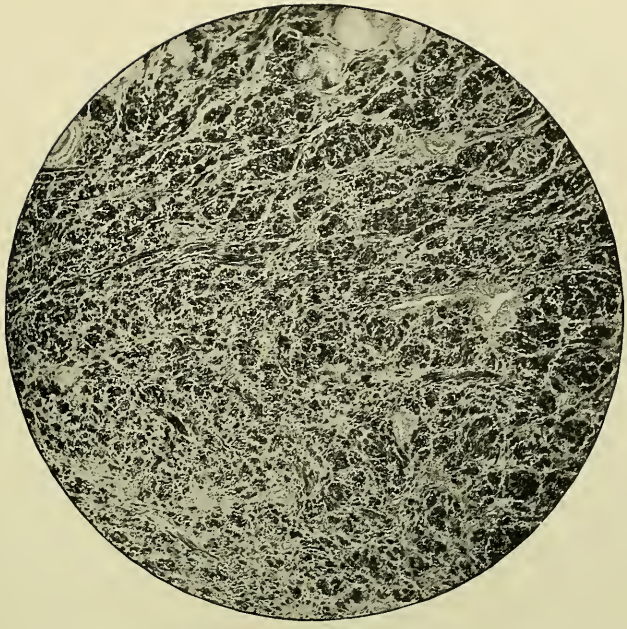

Fig. 15. Showing infiltration of fibrous tissue between the smooth muscle fibres of enlarged prostate.

Fig. 16. Showing diffuse small round-cell infiltration and also arterio-sclerosis in a small vessel.

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practically every field, may be found the remains of at least one or more glands. Such glands, of course, are rarely seen in uterine fibro-myomata.

In those prostatic tumors, which most resemble fibro-myomata (Fig. 15), careful study shows that the distribution of muscle bundles is brought about not by a new growth of them, but rather by subdivision and separation of old bundles due to the ingrowth and expansion of new connective tissue. It has been already shown, also, that the "knotty" form, where small round bodies resembling small fibroids shell out, is always glandular in its origin. Jones (39, p. 240) has never seen pure myoma of the prostate; Virchow (74) says it is very rare, and with this Motz (1, p. 254) agrees. A recent study (3) of one hundred prostates from men dead of prostatism showed fourteen cases said to be adeno-carcinoma. This seems an undue proportion of true malignancy, and so far as present figures go can hardly be taken as an usual percentage.

It is to be concluded, therefore, that true fibro-myoma as well as true adenoma may occur, but that neither is the condition found in the usual benign senile enlargement of the prostate.

3. *Is enlargement of the prostate a senile involution?* The affirmative reply to this inquiry is not to be lightly cast aside. We have seen that there may exist a purely senile atrophy of bladder-wall muscle with a new growth of connective tissue infiltrating the muscles. Such a new growth of connective tissue may take place as part of a purely degenerative process, as is apparently seen in chronic mastitis and in chronic interstitial nephritis. Such connective tissue formation following degeneration is accompanied by the presence of small round mononuclear cells, and such tissue has also its characteristic power to contract and cause constriction. It is impossible, therefore, at present, to exclude senile degeneration as one, at least, of the causes of enlarged prostate.

4. *Is enlargement of the prostate inflammatory in origin?* The best work on this question has been done by Finger (22) and Ciechanowski (1 and 2), and from them I shall transcribe freely.

It has been the custom clinically to separate chronic prostatitis from senile enlargement, the principal ground for this being (*a*) that many individuals suffering from prostatism forget, truly or not, that they have had in the past an inflammatory affection of the urethra or prostate; (*b*) chronic prostatitis may be frequently differentiated in diagnosis from the commoner senile enlargement. This, however, as has been pointed out (1, p. 291), may well be only an example of the remote connection which may exist between the cause and the ultimate appearance of a disease. That is, this latent relation between infection and senile enlargement may be similar to the apparently undoubted connection between syphilis and the later central nervous affections, or the relation between rheumatism and the chronic valvular cardiac disease discovered many years later; (*c*) the objection of Thompson that inflammatory disease should cause shrinking rather than enlargement has already been met by showing that the essential change is a shrinking, but that the shrinking may be about exit ducts and be followed by proximal gland dilatation. It may be, in fact, compared to the constriction of the pylorus, which is followed by dilatation of the stomach. Furthermore, infectious processes may be the basis of increase in volume of tissue, as witness thickenings of the pleura, infectious granulomata, the development of tuberculous tissue, and others. Finger (loc. cit.) concludes that the changes of chronic prostatitis are identical with those of senile enlargement. In both there is a catarrhal gland inflammation, a sub-epithelial, periglandular, round-cell infiltration, and in both the process is in clumps and irregular in distribution. The importance of such differences in intensity and distribution of the inflammation, that is, quantitative differences between prostatitis and enlargement, lies in the fact that they explain differences in clinical symptoms between the two. The amount and intensity of inflammation particularly explains the late and slow onset of enlarged prostate, it being fair to conclude that the latter is always the result of an infection long latent and of such slow onset that the infection may indeed be forgotten. In both processes, described clinically as chronic

prostatitis and enlarged prostate, the beginnings seem always to be in and about the glands and ducts, and not near vessels; in other words, they are both ultimately of urethral and not of hematogenous origin.

The underlying inflammation is, of course, gonorrhœa. That an attack of this infection should be remembered is, as a matter of fact, unneccessary. Patients in whom strictures are found (1, p. 296) will declare they never had gonorrhœa or instrumentation. Many observers (30, 60, 3 quoted by 1) have proved that gonorrhœa may be primary in the prostate, and others (20, 23, 31) have shown that the disease may remain in the posterior urethra for a long time latent and unsuspected. Posterior urethritis is said by some (9) to occur in 93 per cent. of cases with anterior urethritis, and in many of these cases there is a definite prostatitis.

The direct proof of finding gonococci in the sections of enlarged prostate remains unmade. Councilman (14) first demonstrated their presence in the prostate, and others (60) have shown them to be present in prostatic milkings. These instances, however, were in fairly acute inflammations and, if it were difficult here, it is little wonder that they may not have been found in such a chronic and such a mild process as the senile enlargement.

Ciechanowski collects certain indirect facts which tend to confirm belief in the co-relation of gonorrhœa and the enlarged prostate.

1. The frequency of gonorrhœa.

2. The frequency of chronic gonorrhœa in the posterior urethra and prostate, *i.e.*, in 1,070 cases (70) the process was in the deep urethra in 424.

3. The frequency of cystitis; and lastly

4. The only domestic animal that suffers from enlarged prostate is the dog; and the male dog, too, seems to be the only animal that has a true purulent urethritis which is infectious (41).

Kryzysztalowicz (1, p. 299) collected 121 strictures in 99 persons, of whom 60 had suffered from long-standing gonorrhœa, and 5 still had it; ages between forty and sixty years. Most of them first showed demonstrable signs after fifty

years of age. This proves that often many years may elapse after the chronic gonorrhœa before stricture appears, that gonorrhœal infection, in other words, may be latent and forgotten for many years and yet be potent.

And finally, as Ciechanowski (1, p. 300) has made perfectly clear, a characteristic of the advance of a gonorrhœal process is that it is not by continuity but by jumps, and the distribution of diseased foci separately here and there over the prostate is distinctly a feature in senile enlargement.

From this review, therefore, I make the following *conclusions* on the *cause of enlarged prostate:*

1. The underlying cause of the usual form of prostate enlargement, and of certain forms of prostatic atrophy, is a slow formation of new connective tissue, due to infection or to infection aggravating a senile degenerative process.

2. The gonococcus is probably most often the specific infection, because (*a*) of its great frequency; (*b*) other inflammatory causes are not common in the parts in question; (*c.*) a great similarity exists between the histology of gonorrheal processes and those seen in these senile prostates.

3. Neoplasms, fibro-myoma, and adenoma occur, but may be called rare.

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III.

A NOTE ON PLEURISY IN TYPHOID FEVER.

By George G. Sears, M.D.

A report of the following case, whose true character was not suspected until the patient had been under observation for a number of days, may be of interest in calling attention to the probable fact that pleurisy resulting from infection with Eberth's bacillus may be less rare than the published statistics give reason to suppose, but is overlooked because it simulates so closely so-called idopathic pleurisy that it is not given sufficient study to be recognized.

An Italian laborer, twenty years old, was admitted to the First Medical Service of the City Hospital on February 24, 1902. He had been seized fourteen days before with a sharp pain in his right chest, which was followed by cough with frothy expectoration and progressive dyspnea. There was fever and headache. Examination showed the right chest to be well filled with fluid, and 2,000 c.c. of clear serum, with a specific gravity of 1,025, and containing one-half per cent. of albumen, were immediately withdrawn. While only a small portion of the fluid re-collected, the temperature continued high, and the chart strongly suggested the typhoid curve. His mental state was also somewhat typhoidal, but his condition at no time gave cause for special anxiety. The bowels were slightly loose, but the stools were not characteristic. Except for a possible enlargement of the spleen the abdomen showed nothing abnormal. No rose spots were noticed. The Widal test was negative on the thirty-fifth day of the disease, but positive on the thirty-eighth and later. A marked reaction was also obtained with

serum aspirated on the fifty-ninth day. Positive proof of the presence of Eberth's bacillus was lacking, as the effusion was sterile, but strong circumstantial evidence was present in the appearance of the Widal reaction after its previous absence, in our inability to discover tubercle bacilli in the sputum in many examinations, and in the failure of the patient to respond to tuberculin injections. The temperature chart, as convalescence set in, was typical of typhoid.

Although pleurisy has long been one of the well recognized complications of typhoid the direct dependence of one condition on the other has been definitely shown only since the discovery of the bacillus typhosus. It appears however to be one of the rarer complications. Betke [1] found 58 instances among 1,420 patients, Osler [2] met with it 5 times among 389 cases, Gillies [3] 6 times among 249, Ivanoff [1] 11 times in 143, Stritzl [1] 7 times in 122, while A. A. Smith [4] makes no mention of it in an analysis of 87 cases. Remlinger [5] mentions 7 cases as occurring among 1,055 patients. Among 1,065 patients with typhoid fever admitted to the Boston City Hospital since January 1, 1899, it was positively diagnosed but 18 times, yet it is doubtful if this may not understate the true proportion since the difficulty of diagnosis between it and pneumonia, which is often considerable, is greatly increased when dealing with patients too ill to render any assistance to the examiner, while in the cases which develop late and, as usually happens, insidiously, it may easily be overlooked from the disinclination to examine too carefully patients whose condition is critical. A fairer estimate may perhaps be obtained from autopsy records, although the percentage of mortality among the published cases is low. It was present in 6.7 per cent. of 1,073 autopsies recorded by Hölscher, [1] and in 9.5 per cent. of the fatal cases in Betke's series. It occurred three times among 53 autopsies at the Montreal General Hospital.

Although reference is made in these statistics to a considerable number of cases, Remlinger, in 1900, was able to collect from the literature only 31, including 7 of his own. These, with a few overlooked by him, a few which have been published since, and 18 taken from the City Hospital records,

give a total of 55, in many of which, however, a positive demonstration of the presence of the typhoid bacillus was lacking. References to others were found in a somewhat hurried search, but the details were too meagre to make them useful for analysis.

Forty-six of these patients were males, 9 were females, a disproportion for which no explanation is apparent.

It may appear at any time during the course of the disease, but, except as an initial event, it is rare during the first week. It is most common during the later stages of the disease, but may appear as the earliest and most prominent symptom of its onset or be delayed until convalescence seems fully established. It may also occur as a complication of a relapse. It may show itself by the usual symptoms of pleurisy, but more frequently its onset is insidious and its presence is only discovered by physical examination.

Neither side seems especially liable, since out of 54 cases, in which it was definitely localized in the reports, the right and left pleura were each affected 23 times, while in 8 it was double. Twelve of the cases were not aspirated, and the character of the effusion remains in doubt, but as all recovered it was probably serous. Among the other 42 it was serous in 14, purulent in 16 and hemorrhagic in 7. In one case pus was present in one pleura and serum in the other. The process was represented in 4 solely by a friction rub. References were found to other cases in which the effusion was loculated, the pockets containing different forms of exudate. Pneumo-thorax was an occasional though rare occurrence.

Etiologically Eberth's bacillus has been demonstrated as the causative agent in a considerable proportion of cases, and in a few of those in which the the disease was ushered in by marked symptoms of pleurisy the benign course, and the absence of abdominal symptoms, save for a slight gastric disturbance, give reason for questioning if the process was not confined to the pleura; but only two cases were found by the writer in which this was definitely shown, in both of which the infection was secondary to tuberculosis. One was reported by Charrin[6] and Roger, the other by Kelsch.[7] In

the former the typhoid bacillus appeared in pure culture, in the latter tubercle bacilli were also present. Autopsy showed a complete absence of typhoid lesions.

In a smaller proportion of cases pleurisy was the result of a secondary infection with other bacteria, staphylococci or streptococci. In 2 cases in the City Hospital series, not included in the above, an empyema was secondary to abscess of the lung, in one of which the typhoid bacillus was fairly distributed in various organs, but the staphylococcus aureus alone was found in the lung and pleura.

Emboli were demonstrated as the carriers of infection in a few of the cases which developed later, and the proportion in which pleurisy followed phlebitis of the femoral veins or intestinal hemorrhage, showing the depth of the ulcerative process, among the City Hospital cases was quite suggestive of this method of distributing the infection, phlebitis occurring in 3 cases, and hemorrhage in 2, while both were present in 1.

An interesting point in the bacteriology of these cases was the tendency of Eberth's bacilli to disappear from the effusion, which later became sterile.

The Widal reaction was tried with serum drawn from the pleural cavity in a number of cases, with varying results. In some it was negative, while in others a more intense response was obtained than when the blood was used. Achard succeeded by a special process in cultivating the bacilli from serum which gave a very distinct reaction.

The white cells were counted in only a few cases, but these seem to show that their number either remains normal or is but slightly increased, unless the effusion is purulent, when a marked leucocytosis is to be expected.

The character of the effusion, which shows a close relation to the period of the disease in which it develops, has an important bearing on prognosis. None of the initial cases ended fatally, the course being often noticeably mild, while a considerable proportion of the later ones died. None, however, which came on before the twelfth day were either hemorrhagic or purulent. A serous effusion seemed to add but little to the gravity of the original disease, while 2 of the 7 in

which it was hemorrhagic, and 5 of the 17 in which it was purulent, died. Free incision and drainage of an empyema was usually required, yet a few recovered after repeated aspirations, with or without the injection of antiseptics.

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IV.

SOME OBSERVATIONS UPON THE VALUE OF THE PHLORIDZIN TEST FOR ESTIMATING THE FUNCTIONAL CAPACITY OF THE KIDNEYS — RENAL SUFFICIENCY.

By Drs. Francis S. Watson and W. T. Bailey.

The growing realization of the inadequacy of the ordinary methods of urinary analysis for estimating the sufficiency, or the reverse, of the renal function has led, recently, to further efforts to improve them. As a result, there have been brought forward a number of new tests for the purpose, the most noteworthy of which are as follows:

1. Cryoscopy, which is the name given to the test that consists in a comparison of the freezing points of the urine and of the blood of patients whose kidneys are diseased with those of the same fluids of persons whose kidneys are normal.

2. The so-called "Toxic" test (this being more especially addressed to the study of uræmia).

3. The Methyl blue test (by which the permeability, as it is termed, of the kidney is established).

4. The Phloridzin test.

The last of these is the subject of this communication.

The authority of those who are its advocates, and the claims which they make in its behalf, are such, if the latter be substantiated, as to give it a place of great importance amongst the recent advances in clinical medicine. The claim, in brief, is as follows:

That the phloridzin test is a means for estimating the sufficiency of the renal function,* superior to the ordinary

*The term "sufficiency" is used in the sense given it by Sir Andrew Clarke, to indicate the degree of functional working capacity of the kidneys sufficient for the maintenance of bodily well being, not only in ordinary conditions, but also under the strain of surgical operations, accidents, intercurrent illnesses, etc.

methods of urinary analysis hitherto employed for that purpose, and that it is so delicate and accurate as to be a reliable basis for decision with regard to the performance, or not, of such surgical operations as depend for their success in any important degree upon the integrity of the renal function. The most conspicuous example of this class is, of course, that of cases in which nephrectomy is contemplated, and in which it is of the utmost importance to know beforehand whether or no the kidney which is to remain after the operation will be capable of doing the increased amount of work that will be demanded of it. The test in these cases is applied to the urine taken from each kidney séparately by means of uretheral catheterization, or by segregation of the urine in the bladder by Harris' instrument.

The technique of the phloridzin test is exceedingly simple. It consists of the administration of a subcutaneous injection of a sterilized preparation of the drug to which an equal quantity of $Na_2\ co_3$ is added, the latter being used to hold the phloridzin in solution. The dose of phloridzin is five millegrammes for persons of moderate size and weight, and twice this quantity for those of large size and unusual weight. The bladder should be emptied just before the injection is given. Half an hour after the administration of the drug sugar should appear in the urine if there is normal sufficiency of the renal function. Serious disease of the kidneys is indicated if none is present. And if its appearance is delayed, or only a small percentage is found, it is taken as an indication of renal insufficiency. The quantity of sugar in these tests that shall be taken for the standard by which to determine renal sufficiency, or the reverse, is a somewhat arbitrary one. In our own series of observations we took for the point of division between the normal and abnormal performance of the renal function the sugar percentage which stood midway between the average percentage of the cases in which the kidneys were believed to be normal, and the same of those in which renal disease was known to be present, which we found to be .30 of one per cent., all percentages of sugar falling below this number being taken as an indication of renal insufficiency in proportion to the degree in which they were less.

The following things may be taken as clearly established with regard to phloridzin : It is a drug from which glucose is separated in the course of its passage through the body, as is shown by the presence of sugar in the urine subsequent to its administration.

The phloridzin test shows that both kidneys, under normal conditions, have an equal functional capability.

Phloridzin is perfectly harmless.

In addition to the above, it is further asserted, though less well proved, that the separation of sugar from phloridzin takes place in the epithelial structure of the glomeruli and tubules of the renal cortex *and in no other part of the body.*

That much of the value of the test depends upon the truth of this statement is obvious when we remember the twofold nature of the renal function.

There are, as we know, two distinct factors invloved in it, both of which are necessary to its proper performance. These are, *first*, the integrity of the epithelial structure, just mentioned, without which it cannot effect chemical changes in certain substances and deliver them to the urine in the form of waste products, such as urea, etc., and, *second*, the normal degree of permeability of the membranes which interpose between the blood and the urine in the renal parenchyma.

Now, if we find sugar in the urine according to the requirements of the test, and if it is true that the separation of sugar from phloridzin does take place by virtue of the first of these two factors, we must necessarily conclude that the second of them has been properly performed also, since the drug could not have reached the epithelial structure without passing through the membranes referred to and had the latter not had normal permeability. Thus phloridzin tests both functional factors of the kidneys, and is therefore superior to other methods, such as the methyl blue, which gives information only with regard to one of them, namely, permeability.

There is one experiment, however, which, so far as one experiment can do so, speaks strongly against the exclusive power of the renal epithelium to separate sugar from the drug. It is one undertaken by Domenicis.

He gave a known dose of phloridzin to a dog, and found, within a specified time afterward, a certain quantity of sugar in the blood. Some days later he removed both kidneys from the animal, and repeated the test as before. He then found a larger quantity of sugar in the blood than on the first occasion, conclusively showing that the separation of sugar from the drug was capable of being accomplished by some part of the organism other than the kidneys, since, in his last test, there were no kidneys present by which it could have been done.

What has been said thus far is a brief résumé of the experience of other observers up to the present time. What follows has to do with our own observations.

In the course of one term of hospital service the number of cases in which the test could be applied to determine the condition of each kidney, independently — more especially in cases of contemplated nephrectomy — is too few to make that particular investigation seem worth while, but it was thought that the value of the test could be estimated without confining it to this special class.

It also occurred to the writer that it would be of interest to use the test for the purpose of estimating the effect of ether anæsthesia upon the renal function, and to compare the results with those which have been already arrived at through a thorough study of the same subject by means of the ordinary methods of urinary analysis. The latter, it will be remembered, showed that both ether and chloroform anæsthesia are followed, in a large proportion of cases, by the appearance *de novo*, in the urine, of casts, albumen, and other signs of renal disturbance, which have been assumed to be evidence of the injurious influence of these anæsthetics upon the kidneys and their function.

In addition to this, I sought to utilize the test to determine the following points:

1. The average amount of sugar eliminated by the kidneys within a specified time after phloridzin administration.
2. The rate of the sugar excretion.
3. The value of phloridzin test as compared with that of the ordinary methods of urinary examination, and finally to

note such matters of interest as might be furnished by individual cases.

Our observations extended over a period of seven months, and would not have been possible for me to carry out without the disinterested collaboration of Dr. Bailey, who devoted a great deal of time to the detail of the work. I am also greatly indebted to my colleague, Dr. H. L. Burrell, who gave every facility that his wards afforded during the earlier experiments, and to the surgical house-officers, who took much pains to ensure their success.

The following general rules were observed with regard to the manner of doing the tests:

1. First, a routine test for sugar was made in all cases, in order to be sure that it was not already present in the urine.

2. The bladder was emptied just before the phloridzin injection was given.

3. The test specimens of urine were drawn with a catheter.

4. Two specimens were taken in as many cases as possible, both before the patients were etherized and during anæsthesia. In the latter, the phloridzin injection was given just as anæsthesia was begun. The first specimen was taken at the end of the first half-hour, and the second at the end of the first hour after the administration of the drug. Thus in the full tests, five specimens were examined in each case. The tests which were made prior to etherization we called the *preliminary*, and those during anæsthesia the *ether phloridzins*.

5. Twenty cc. of urine was used for each test.

The total number of patients tested was seventy. We have formulated our observations with reference to the different points to be studied.

The average quantity of sugar and its rate of elimination, when the kidneys are normal, were established by calculating the average percentages found in both the half-hour and the hour tests made in the same cases, the patients not being anæsthetized. Of these there were forty-six. The average sugar percentages in the half-hour and hour tests were

respectively .44% and .38%. *The rate* of sugar elimination is seen by comparing these figures, which show that the quantity of sugar eliminated in the first half-hour was greater than that in the second half-hour by .06%.

The effect of ether anæsthesia was determined by comparing the quantities of sugar in the half-hour and hour urines of the ether phloridzins with the same in the preliminaries, where both were taken in the same cases, and with patients not having kidney disease. The number available for this purpose was twenty.

The half-hour and hour sugar percentages of the ether phloridzins in these were respectively .68% and .53% as compared with .42% and .36% in the preliminaries, from which it will be seen that the effect of ether anæsthesia is to increase both the rate and the quantity of sugar elimination. The rate is shown by the relatively as well as actually larger quantity of sugar in the first half-hour than in the second, as compared with that of the corresponding tests in the preliminaries. The difference in the ether phloridzins being .15%, that in the preliminaries .06%, or less than half as much.

It is to be regretted that we were unable to examine the urines of these patients for casts, albumen, urea, etc., while the kidneys were under the same degree of anæsthesia as they were from the phloridzin tests. The failure to do so leaves the experiment defective, in that we cannot say what proportion, if any, of the cases of these etherized patients may have shown the evidences of renal disturbance by the ordinary methods of analysis of the urines.

CASES OF RENAL DISEASE.

Our next object was to test the value of phloridzin in estimating the condition of the renal function in disease of the kidneys, which we judged by applying the test to the cases in which the latter was known to exist, it having been demonstrated by the ordinary method of analysis. Of these there were thirteen. One of them, in which the sugar percentage was exceedingly high, 1.30%, was omitted from the

calculation, because to include it would give an erroneous impression with regard to the sugar elimination of the rest. The average sugar percentages in the half-hour and hour preliminary phloridzins in the remaining twelve cases were respectively, .21% and .20%, those of the corresponding ether phloridzins were .22% and .20%.

These general averages present a striking and significant contrast to the percentages of sugar shown by the same tests in the cases in which the kidneys were normal. The latter having almost twice as much sugar in the urines of their preliminary, and three times as much in their ether phloridzins.

Another noticeable point in these cases is the very slight difference in the amounts of sugar eliminated in the first and second half-hours of both the preliminary and ether phloridzins, as compared with those in which there was no renal disease. Thus for the former, the half-hour and hour preliminaries show a difference of only .01%, and in the ether phloridzins of only .02%, while the same tests in the latter give respectively differences of .06% and .15%.

This failure of ether anæsthesia to increase the elimination of sugar in cases in which the kidneys are diseased, seems to the writer to be the strongest testimony furnished by our observations in support of the delicacy of the phloridzin test.

The cases of renal disease are given in detail in the following table:

	Prelim. Phlorid. ¾ hour.	Ether Ph. ¾ hour.
1. Prostatic Hypertrophy. Chronic interstitial nephritis. Aortic valvular disease of heart. Death. Prostatectomy. Death due in part to renal failure	.12%	.10%
2. Urethral Stricture. Chronic nephritis. Operation. Recovery. Serious constitutional disturbance following operation. Urinary suppression threatened	.22%	.13%

3.	Urethral stricture. Chronic nephritis. (?) Slight constitutional disturbance, post operative. Recovery . .	.40%	.55%
4.	Unilateral calculus pyelonephritis. Nephrolithotomy. Uneventful convalescence	.16%	.13%
5.	Tubal pregnancy. Moderate parenchymatous nephritis. Operation. Death. General peritonitis . . .	.22%	.18%
6.	Cervical adenitis. Moderate parenchymatous nephritis. Operation. Uneventful recovery	.25%	.40%
7.	Axillary abscess. Chronic nephritis. Operation. Slow recovery . .	.13%	.13%
8.	Fractured femur. Chronic nephritis	.15%	.17%
	Total per cent.	1.65%	1.79%
	Average per cent	.20% +	.22%

In addition to these eight cases in which the *preliminary and ether tests* were made, there were four other renal cases in which *only preliminary phloridzins* were made, as follows:

		Preliminary Phlor.	
1.	Amyloid disease of kidneys . . .	.22%	
2.	Kidney fixed in abnormal position, by adhesions	.13%	
	In this case after nephrectomy one kidney alone gave sugar per cent. of . .		.30%
3.	Ruptured kidney, urine from other kidney alone. Nephrectomy. Death,	48%	
4.	Unilateral pyonephrosis. Sugar per cent. of urine from both kidneys was	.22%	
	Total per cent.	1.05%	
	Average per cent. . . .	.26%	

INDICATIONS FURNISHED BY THE STUDY OF SPECIAL CASES.

The general results of our observations, which have thus far occupied us, are almost wholly in confirmation of the

claims made for the value of the phloridzin test. When we come to study it with relation to individual cases, however, certain contradictions and defects become apparent. These are as follows:

First, a group of seven cases in which not the slightest evidence of renal disease could be found by any of the usual methods of examination, but in which the phloridzins gave very low sugar percentages in every instance, as will be seen in the following table.

Cases in which renal disease or defective renal function could not be demonstrated by any of the usual methods of examination:

	Half-hour phloridzins.	Hour phloridzins.	Disease.
Case 1	.13%	.14%	Hernia.
" 2	.16%	.14%	Osteomyelitis.
" 3	.13%	.11%	Hydrocele.
" 4	.13%	.11%	Sprained ankle.
" 5	.13%	.15%	Fractured patella.
" 6	.16%	.13%	Cellulitis of neck.
" 7	.18%	.16%	Cervical abscess.

None of these cases were, it is true, submitted to the practical test of operation upon the urinary organs, but the entire absence of evidence of renal disease or impaired function, that the ordinary methods of examination showed, is enough to make one suspicious of the value of the phloridzins and their findings in these cases.

Six further cases furnish contradictory evidence as to the worth of the phloridzin test, two of them being in its favor and four speaking against it. Two of them also are examples of the reverse of the seven cases given above, in that they show *high sugar percentages* in cases in which kidney disease was present, both patients being the subjects of chronic interstitial nephritis, one of them having a sugar percentage of 1.30% and the other of .47% for single three-quarter hour tests in each case.

Two others were old men suffering from serious forms of prostatic hypertrophy. The sugar percentages of the pre-

liminary tests were low in both, being respectively .12% and .15%. In the former there was slight evidence of renal insufficiency shown by the ordinary urinary analyses; in the latter there was none forthcoming. The result of prostatectomy in the first was death with marked uræmic symptoms, and of the second, recovery without the least disturbance of the renal function, yet the phloridzin test had given almost equal warning of trouble to be expected in both. There was a post-operative phloridzin test made in each of these cases, however, in which the results differed widely, the patient who died showing a still lower sugar percentage than in the preliminary tests, namely, .10%, while the patient that lived had a post-operative sugar percentage of 2.00%, the highest in the whole series of our cases. There was nothing to explain this very high figure.

Of the two final cases, one showed a very low percentage of sugar .09%, which proved a sound guide, in that the patient had very serious symptoms after being operated upon for relief of urethral stricture, threatened suppression of urine being prominent amongst them. The second, on the contrary, made a perfectly uninterrupted convalescence after a nephrectomy, although there had been here also a very low sugar percentage shown by the preliminary phloridzins, it being .13% in this instance. There was one peculiarly interesting feature in this last case, which was, that the single kidney remaining after the operation secreted within a very short time subsequently a much larger quantity of sugar than the two kidneys together had done prior to operation. This might be taken as another example of those which have been cited elsewhere as showing the inhibitory influence upon the functional activity of one kidney exercised by injury or disease of its fellow. The case was one in which the left kidney was bound down by dense adhesions in an abnormal position, and in which the patient was practically disabled by the constant renal pain due to this condition. The ordinary methods of urinary analysis showed no evidence of renal disease in either of the above cases.

Here, then, are eleven cases out of the seventy in which the phloridzin tests apparently furnished unreliable indications.

There were but three cases in which the urine of either kidney was tested independently of the other.

The first is that already described, in which one kidney was fixed in an abnormal position.

The second is one in which the writer performed lumbar nephrotomy six years ago for the relief of acute pyonephrosis. The urine from that kidney has been wholly discharged through a fistulous opening in the loin ever since. Its function, judged by the ordinary methods of examination, has been usefully though imperfectly carried on up to the present time, there being a fair amount of the urinary solid constituents excreted. The urine coming from the other kidney has been normal, showing that the organ has compensated well for the damage done to the other. The phloridzin tests in this case confirmed the testimony given by the usual methods with regard to the sound kidney, showing a good sugar percentage in both the half-hour and hour tests. But in the case of the damaged kidney they indicated a graver condition than was suggested by the usual urinary analysis. In the latter instance, I believe, from what I know of the condition of the kidney, that the phloridzin is nearer the truth than the other. The phloridzin half-hour test showed no sugar at all in the urine from the damaged kidney and only .25% in the hour test.

The last case of these three is that of a man with a ruptured left kidney, which was removed by abdominal nephrectomy, the patient dying four days later. In this case the ureter of the injured kidney was torn across so that no urine from it reached the bladder. That which came from the other kidney was tested after being drawn from the bladder, and showed a sugar percentage of .48%, the point of interest here being that the function of the sound organ was apparently undisturbed by the destruction of its fellow or the shock of the system, which was severe.

CONCLUSIONS.

The conclusions that seem warranted by the results of these observations are as follows:

1. That the average quantity of sugar eliminated in the

first half-hour after the administration of the drug subcutaneously in the doses stated, and when the kidneys are normal, is about .45%, and that the first half-hour's elimination is greater than the second half-hour's by about .06%.

2. When renal disease exists the first half-hour's quantity of sugar eliminated is, for a series of cases, about one-half as much as that when the kidneys are normal, and there is but very little more in the first half-hour than in the second.

3. The effect of ether anæsthesia is to stimulate the kidneys to greater functional activity, but the renal function is not, if judged by the phloridzin test, in any way impaired by the anæsthesia. This is true of the kidneys when normal only; when they are diseased ether fails to stimulate their function, and there is, moreover, relatively much less sugar eliminated in the first than in the second half-hour after phloridzin has been given than is the case when the kidneys are sound.

If judged by the average results of a series of cases taken collectively, the phloridzin test gives accurate indications of the condition of the renal function and of the existence of renal disease. In a certain number of individual cases it even appears to be more delicate and discriminating than the ordinary urinary tests, but the relatively large number of instances in this series of observations in which the reverse of this is true show it to be too variable to be trustworthy, or to incline us to urge its adoption in preference to the former methods of urinary analysis for estimating the functional capacity of the kidneys.

V.

A CASE OF POISONING BY THALLIUM.

By William N. Bullard, M.D.

The metal thallium was discovered by Crookes in 1861, and since then we have obtained some knowledge through experiment of its toxic effect on animals. Cases of poisoning in man are, however, not common.

POISONING IN ANIMALS.

The principal symptoms found in animals have been general listlessness, loss of appetite, nausea and vomiting, diarrhœa with dejections often tinged with blood, and intestinal pain, sometimes severe. There is slowing of the respiration and dyspnœa. The chief action, however, seems to be upon the heart, on which it is supposed to act as a direct poison. It causes slowing and weakening of the heart's action, and the heart stops in diastole.

In addition to these symptoms it produces general tremor of the limbs and a marked incoördination, usually more evident in the hinder extremities. Luck notes a marked swaying on standing, and a slow, uncertain, swaying gait. Richet found in chronic poisoning a generalized muscular atrophy, not a simple leanness, but an almost total wasting of different muscles.

Curci states that thallium does not act on the nervous system of animals, but that it is exclusively a muscular agent.

Both Buschke, in 1900, and Bettmann, 1901–1902, succeeded in producing alopecia in animals with acetate of thallium.

The only definite abnormal conditions found pathologically were swelling, congestion, and small hemorrhages of the gastric and intestinal mucous membrane; sometimes congestion and small hemorrhages in the lungs, and mild parenchymatous nephritis. The heart is usually stopped in diastole, and its right cavities are dilated.

POISONING IN MAN.

The instances of this are rare. Crookes states that he swallowed 0.065–0.130 grammes of thallium salt without any effect. Pozzi and Courtade employed the iodide of thallium in doses of 0.010 gramme daily in certain cases of syphilis with favorable results; as unpleasant symptoms were mentioned pain in the stomach and vomiting. They also found in a few cases that the gums were painful and swollen, and that there was a blue line at their junction with the teeth. I have not found these latter symptoms elsewhere mentioned.

In 1898 Combemale advocated the use of the acetate of thallium in the profuse sweats of pulmonary tuberculosis, and found it most efficient for this purpose. The ordinary dose was ten centigrammes, the maximum twenty centigrammes. He never observed any evidences of toxic action while the drug was being taken, but noticed a falling of the hair later. This was said to be total and extraordinarily rapid. Huchard, writing a report on this paper for the Académie de Médécine, states that he had tried this drug on two patients in the last stages of phthisis. They were attacked with very sharp pains in the lower extremities during the time thallium was taken, and he concluded that these pains could not have been due to the phthisis, because they ceased immediately on stopping the thallium.

Jeanselme in the same year reported a case in which a woman, fifty-nine years old, was given cachets each containing three centigrammes (0.03) of acetate of thallium for profuse sweats. In the course of three days she took nine cachets, 0.27 centigrammes, when the medicine was stopped on account of diminution of the sweats and because of the occurrence of rather sharp pains in the abdominal and lumbar

regions. Fifteen days later her hair suddenly began to come out in masses, and she lost about one-third of it.

Guinard also in 1898 reports loss of hair after nine pills of 0.05 centigrammes, of acetate of thallium.

It was thus quickly established that thallium, even when given in small doses, was liable to cause a loss of hair or alopecia in men. This usually occurred some days after the administration of the drug had ceased.

It was found that the loss of hair produced by the administration of thallium was so serious that the use of the drug has been practically abandoned, although it proved a most efficient agent for sweats.

I have been unable to find any further reports of toxic symptoms in man from this drug.

My case was that of a gentleman, twenty-seven years old, a physician, who was admitted to the hospital as a private patient of Dr. V. Y. Bowditch, and whom I saw in consultation a number of times. He had taken sulphate of thallium for purposes of experiment, "not over eight grains in all, and probably not over half a grain at a time; four grains is probably nearer the exact amount." Later he writes: "From half a grain to a grain was taken at a time, and the first time was taken perhaps every other day for three or four doses. Then there was an interval of about two months, then three or four doses were taken again in the same manner. Nothing was noticed after the first lot was taken, but within two or three days after the last dose the numbness in the toes and finger tips was noticed."

On admission to the hospital he gave the following account of his symptoms: Three weeks ago slight diarrhœa, which readily yielded to treatment, and at the same time numbness in the toes of both feet and the fingers of both hands, which, in the course of two or three days, extended up the lower extremities and involved the lower abdomen, the perineum, the inside of the thighs and the inner surface of the legs, being less marked over the tibiæ. The nerves of the lower extremities were painful, and there was considerable weakness and partial paralysis of the lower extremities. Almost from the onset he was confined to bed or to a

wheel chair. He grew worse from the first seven to ten days; then his condition became stationary, and now he considers himself slightly better.

On admission the balls of his feet were exquisitely tender, and there was some tenderness over the whole metatarsal region of both feet and slight tenderness along the inner border of each tibia. There was loss of the power of motion of both big toes, which he could ordinarily move freely. The other toes could be moved without pain.

All other movements of the lower extremities were present, though weak. There was some wasting of the lower extremities, and there was a diminution of sensation over all the toes. The muscles of the lower extremities were lax. Knee jerks lively. Plantar reflexes not obtained. No ankle clonus. There was also some diminution of sensation over the fingers. The internal organs were all natural, and no other abnormal signs were found. The right pupil was slightly larger than the left, but he states that this had been so since scarlet fever, when he was five or six years old. No blue line was found on the teeth or gums.

The physical examination a few days later gave similar results. Has but little power of motion in lower extremities. Cannot move the big toes, but can now move the fifth toes, which he formerly could not do. There is now diminution of sensation over the external border of both feet and the internal third of the right leg. The most tender points now are on the soles of the feet, near the middle of the metatarsi. Knee jerks increased. No clonus.

Eighth day after entrance to the hospital it is noted that he has had for some days considerable difficulty in sleeping. The numbness has now extended so as to involve both knees. He has troublesome twitching pains in both legs and in feet.

Ten days later (eighteen days after admission). Improving. Limbs pain him much less. Sleeps better.

Twenty-sixth day after admission. Great improvement. Able to be up and out in wheel chair. Has had electricity since last report.

A week later we find that traces of lead were discovered

in the urine; also "$\frac{1}{300}$ to $\frac{1}{400}$ m'grms. of arsenic, probably too slight to account for symptoms."

Thirty-third day after admission. Discharged.

During his stay in the hospital the urine was frequently examined; it showed at first slight traces of albumen, which gradually disappeared. On the day of entrance the condition was as follows: Pale, acid, sp. gr. 1,016, slightest possible trace of albumen. No sugar. Occasional abnormal globules and degenerated small renal cells and leucocytes.

Twelve days later. High color. Trace of albumen. Much pus, mostly in clumps. Many normal red corpuscles, some abnormal. Occasional small and large round degenerated cells. Calcic phosphate crystals.

Ten days later it was normal, acid 1,024.

The treatment, besides rest in bed and dry heat to his limbs, consisted only in iodide of potassium in small doses. Later, when the pains had ceased, electricity was applied to the lower extremities.

About three months after the onset of the illness the patient wrote: "Have resumed work, but am not absolutely well. Played golf yesterday without discredit. Am getting used to the cold weather. *My hair all came out.*"

Remarks.— The condition here was plainly a multiple neuritis. It followed the usual course of this affection, and the patient eventually recovered completely.

I do not consider the report of the existence of lead in the urine important. I cannot now determine with certainty by whom the examination was made or what tests were used. It is possible that the tests were really given by thallium. The patient lived in a large institution, where many other persons were exposed to essentially the same conditions as himself, none of whom showed any signs of lead poisoning. On the other hand, the source of the thallium was known, and the symptoms occurred immediately after its ingestion.

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VI.

SIX CASES OF RUPTURE OF THE INTESTINES, WITH FOUR RECOVERIES.

By F. B. Lund, M.D., E. H. Nichols, M.D., and John T. Bottomley, M.D.

The following cases of rupture of the intestine which have occurred during the past year are reported simply to emphasize the value of early exploratory operation in cases of abdominal injury.

Case 1. — Dr. Lund. Rupture of intestine by fall from bridge. Intestinal resection after twenty hours. Death.

M. P., a boy, seven years of age, was brought to the City Hospital on July 20, 1901, at 1 P.M. The mother said that at 5 o'clock the night before the boy ran into the house, crying with severe abdominal pain, and said that he had fallen from a bridge. He vomited once, had a chill, and in the evening became feverish. He slept fairly well, and the next morning was able to get up for water. The pain seemed less severe, but the abdomen was tender. The bowels did not move. He was seen by a physician, who ordered his removal to the hospital.

When seen at the hospital the boy lay with his limbs drawn up, his face was pale, expression anxious, tongue dry, and feet and hands cold. Pulse, 140, very weak and compressible. Temperature, 99.5°. The abdomen was not distended. There was ecchymosis over the crest of the left ilium. The abdominal walls were rigid on palpation, especially on the left side, where there was marked tenderness.

Under ether, an incision was made over the outer border of the left rectus, just below the umbilicus. On opening the peritoneum there was an escape of dark brown serum, containing a few flakes of fibrin. The intestines on the left side were injected, and presented flakes of fibrin on the surface. On drawing them out through the wound a coil was found in the left hypochondrium, wrapped up in omentum, which was torn from its mesentery for about three inches, and in its centre a large perforation. It was denuded of peritoneum. The damaged portion of intestine was quickly excised, and the ends brought together by a Murphy button, reinforced by a few sutures of fine silk. The torn mesentery was united by two silk sutures.

The boy was in severe shock and pulseless during the latter half of the operation, which took thirty minutes. The abdomen was washed out and drained with odoform gauze, and the wound was closed with sutures of silk worm gut. The drainage was carried into the left hypochondrium and into the pelvis.

The boy was stimulated freely, but did not rally, and died two and a half hours after the operation.

Case 2. — Dr. Lund. Rupture of intestine, probably due to the kick of a horse. Operation sixteen hours after accident. Recovery.

A. C., forty-six years old, was seen by the writer, in consultation with Dr. A. B. Brown of Newburyport and Dr. Spaulding of Salisbury, at 9 A.M. on May 6, 1902. At 4.30 the previous afternoon he was driving, when his horse became frightened and he was thrown from his carriage, striking his abdomen, he thinks, against the whiffletree, and being kicked in the shin, and, possibly, in the abdomen, by the horse. When seen by Dr. Spaulding, at about 7 P.M., he had vomited, was in a state of moderate shock, pulse 100, and of good quality. There was a small red spot on the abdomen, about two inches to the right of the umbilicus, and distinct tenderness and spasm on pressure over the right side of the abdomen. Dr. Spaulding suspected rupture of the intestine, and advised calling a surgeon at once. During the night he vomited three times, and the bowels could not be made to move by enemata.

In the morning, when seen by Dr. Brown and the writer, he was found to be a man of good strength and nutrition. He was lying in bed, feeling well, as he said, facies excellent, pulse, 100, and rather compressible, temperature, 100°. On the left shin there was a wound, laying bare the tibia, evidently made by the shoe of a horse. On the right side of the abdomen was a small abrasion, as described above. On pressure in the right lumbar region there was moderate tenderness and slight spasm of the muscles. Exploratory laparotomy was advised, diagnosis of probable rupture of the intestine being made.

Under ether an incision three inches long was made through the outer part of the right rectus, the centre being just below the umbilicus. On opening the abdomen there was an escape of turbid serum. Coils of intestine below the incision were injected and coated with flakes of fibrin. On withdrawing several coils from the abdomen and following up the intestine where the fibrin flakes became thickest, a perforation about the size of a lead pencil was found, which was partly closed by the pouting of the mucous membrane through it. This perforation was quickly closed with interrupted sutures of fine silk. The greater part of the small intestine was removed from the abdomen in the search for further perforations, but none were found. The peritonitis was found to involve the pelvis and the right hypochondriac region. The left side of the abdomen and the left hypochondrium were apparently free from inflammation. A considerable amount of turbid serum was washed out of the pelvis and out of the right hypochondrium. After thorough flushing of the entire abdomen the intestines were returned and the wound sutured. A glass tube and two gauze wicks were passed into the pelvis through the lower end of the incision and a gauze drain passed from its upper angle to the bottom of the right hypochondrium.

The patient left the table in excellent condition. The next morning his temperature was normal, and pulse, 78, but up to 4 P.M. he vomited frequently. The following day the temperature was normal, and the pulse, 82, and of good quality. There was free drainage through the wound. The bowels moved on the second day as a result of enemata. On the third day he had another attack of vomiting, which was relieved by enemata. The movements passed on that day contained peanuts, which he was eating at the time of the accident. The drains were removed on the sixth day after the operation, and two weeks after the operation the wound was healed, except for two small granulating areas where the drains had been taken out.

The patient gradually recovered his strength, and when seen by the writer in August was in good health, except for a slight hernia through the wound, which he was advised to have operated upon.

Case 3.—Dr. Lund. Rupture of the intestine by kick of a horse. Operation nine hours after accident. Recovery.

F. J., aged twenty-eight years, a teamster, was admitted to the Relief Station at 1 o'clock on September 24, 1902. The patient stated that he was kicked in the abdomen by a horse about 6 A.M. on the date mentioned above. He was thrown about ten feet and was unable to rise. At time of the injury the patient had a right inguinal hernia, probably containing a loop of intestine. One of the horse's hoofs struck over the sac and the other struck the right lumbar region. No vomiting at any time since the injury, only slight nausea at times. The accident happened at Weston, Mass. A physician advised transfer to city. He was brought to Boston by train on a stretcher.

The patient was well developed and nourished. Lips pale. Cheeks looked drawn, pinched and pale. Temperature, 98.6. Pulse, 68. Passed urine, which contained no free blood. An enema produced a good movement of the bowels. Chest negative. Abdomen full, but not distended. Some muscular spasm and tenderness in lower half of abdomen, most marked in median line. Pressure over bladder and kidneys did not cause special pain. Point of most tenderness over right external ring, where there appeared considerable swelling apparently connected with hernial sac.

Ether and operation advised and accepted at 3 o'clock. An oblique incision was made over the inguinal canal and tissue about spermatic cord, which were œdematous and ecchymotic. On division of the tissue, the hernial sac was exposed and incised and found to contain clear fluid. Then the aponeurosis of the external oblique was slit up over sac and incision carried up to internal ring. Pressure on abdomen caused extrusion of turbid light brown fluid from abdominal cavity. Incision was therefore carried up through external and internal oblique and transversalis muscles, a distance of two inches, considerable turbid fluid escaping from abdomen. Numerous coils of small intestine, some distended and some collapsed, deeply injected, and partly covered with delicate flakes of fibrin were found. A loop of intestine was drawn out and, proceeding in direction where fibrin was thickest, a laceration three-fourths of an inch long was found opposite mesenteric border, crossing intestine obliquely. Fibrin was removed from the edges of the

laceration and it was closed as quickly as possible with a double row of continuous fine silk sutures. There were several deeply ecchymotic spots in the neighborhood, along the mesenteric border of bowel, in one of which it looked as though perforation were imminent. This spot, therefore, was inverted and sewed with interrupted Lembert sutures. Another spot, where the integrity of the intestines appeared to be dubious, was also inverted.

Several contusions of the mesentery were noted. In the area exposed were four or five enlarged mesenteric glands, over the largest of which (size of a walnut) was a bright red ecchymosis, showiug that this gland and mesentery must have been sharply compressed. All the intestinal coils in the neighborhood removed from abdomen for exploration for further injury, and on being found intact they were thoroughly irrigated with salt solution and returned to the abdominal cavity after thorough irrigation of the latter.

The upper part of the wound was closed with four silkworm-gut sutures. Two gauze wicks were placed for drainage about a glass tube which ran into the pelvis, and one wick was placed a little higher up.

The convalescence of this patient up to the present time (Oct. 1) has been uninterruptedly good. The bowels moved on the second day, and the gauze was removed on the fifth day. There was some swelling of the scrotum, probably due to infection of the hernial sac with intestinal contents. The operation for radical cure of the hernia is in contemplation. The incision was made into the hernial sac in this case for the reason that this was the known position of the blow upon the abdominal wall. In the writer's two cases reported above the incision, instead of being made in the median line, was made over the site of the injury for the following reason: In a case (previously reported)* of rupture of intestine by blow of a board thrown off from a circular saw, where the injury was in the left groin, the writer made an incision in the median line. The rupture of the intestine was found and sutured, but the patient died on the tenth day from the rupture of a subperitoneal-hæmatoma in the left groin into the abdominal cavity. This hæmatoma was probably infected with fæcal contents at the time of the accident, and was not found by the writer at the operation. It gradually became purulent, and was the starting point of an abscess which discharged into the peritoneal cavity. In case the incision had been made over the site of the injury, this hæmatoma would probably have been found and evacuated. Its exact position was between the peritoneum and the anterior pelvic wall formed by the ramus of the pubes, ramus of the ischium, and obturator internus muscle. For the reason that the greatest injury is apt to be located under the seat of the blow, and that concomitant injuries, such as hæmatomata, may be here best dealt with, the writer is in favor of incising the abdomen as nearly as possible over the site of the injury, even if another incision has to be made in the median line in order to drain the pelvis.

In the third case, the tenderness was very slight, there had been no vomiting, and the bowels had moved freely as the result of enemata,

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but the condition of things disclosed at operation would have rapidly proved fatal. There can be no question in this class of cases, wherever there is any suspicion of rupture of the intestine, that it is far safer to operate at once than to pursue a waiting policy, which will inevitably be attended with an unnecessarily high mortality.

Case 4. — Dr. Nichols. Rupture of intestine by kick of horse. Operation three hours after injury. Recovery.

A. A., male, forty-two years, married, teamster. Admitted to the Relief Station of the Boston City Hospital, August 20, 1902.

History. — Between one and two o'clock the day of admission, patient ate his dinner of meat, potato and cabbage. At half-past five P.M. was kicked in the "pit of the stomach" by a horse. He was standing four or five feet from the horse's heels at the time. He was knocked down, and "lost himself" for an instant. The immediate pain was severe, but not so extreme as it became later. He at once was nauseated, and had attacks of retching. The attacks came on at intervals of fifteen minutes, and during the attacks some mucous mixed with bright blood were raised, about a cupful in all. He was brought to Relief Station and seen at 8.30 P.M., just three hours after the accident.

Physical Examination. — Patient was a large man, about 180 pounds in weight, rather thin and muscular. He was very restless, but perfectly clear mentally. He lay upon his back, with legs fully extended. The face was somewhat pale, with a dusky tinge, and looked rather pinched. Tongue was clean and moist. Temperature was 99.6° F. Pulse was 100, of fair strength, regular, but quite compressible. The chest was not examined. On the right lower abdominal wall was a very slight redness and abrasion of the abdominal wall. There was no distention of the abdominal wall, but if anything a slight retraction. He complained of general abdominal pain. On palpation there was general abdominal tenderness, not, however, extreme, and no point of marked local tenderness. The abdominal muscles were literally as "rigid as a board" everywhere. On percussion there seemed to be slight dulness in the right iliac region. The diagnosis was made of rupture of the intestine, and operation advised.

The operation was begun just three hours after the injury. Under ether an incision two and one-half inches long was made in the middle line half-way between the umbilicus and the pubes. When the peritoneum was opened there was an immediate escape of considerable cloudy serous fluid, tinged with blood. The intestine beneath the incision was very much injected, and covered in places with small amounts of fibrin. Following up the injected fold an oval perforation one-half an inch long was found in the ileum, opposite the mesenteric attachment, eighteen inches from the ileo-coecal valve, from which fecal contents escaped. The incision was closed with a continuous right-angled silk suture, reinforced with interrupted silk sutures. A hasty examination of the intestine showed no further rupture. The peritoneal cavity was washed out with very large amounts of normal salt solution. During the process much fecal matter was found in the true pelvis. Irrigation was continued until the fluid was perfectly clear. Gauze drains were put into each flank, under the liver and into the pelvis, with a glass drainage tube into the pelvis.

The condition of the patient was good throughout the operation. On recovering from ether the patient vomited twice, and at no other time during convalescence. The bowels were moved by a high enema the next day. For the first three days nothing was given by mouth, but feeding was by enemata. On the fourth day very small amounts of beef juice and tea were given by mouth. On the seventh day egg-nog and milk were given by mouth, and on the tenth day soft solids. There was no distention, and tenderness was always slight and rapidly disappeared. The drains were all removed by the thirteenth day. The patient sat up at the end of three weeks, and left the hospital wearing a fitted swathe on the twenty-third day. The only feature which caused anxiety during the convalescence was an occasional attack of moderate dyspnœa, for which no cause ever was found.

Case 5. — Dr. Bottomley.

A fisherman about forty years old was brought to the Relief Station in a police ambulance, and gave the following history: Six hours before, while watching a fight, he had been struck on the back "just below the left shoulder-blade" by the shaft of an approaching patrol wagon, and had been knocked down. He felt faint for a short time, but did not lose consciousness. Severe abdominal pain, chiefly above the umbilicus, followed the blow. Arising soon, he walked about for a while, took a carriage to the ferry, a short distance away, and after crossing walked three quarters of a mile to his home. A cup of tea which he took there brought on the first attack of vomiting. His abdominal pain increased in severity and he summoned his physician, who ascribed the severe pain to a distended bladder, which he found, and for the relief of which he advised the patient's removal to a hospital. There had been but one attack of vomiting since the accident, and at the time of his coming to the hospital, the only subjective symptom was severe abdominal pain.

He was a well-built, well-nourished man, and while his pulse was only 92, its character was not satisfactory; it was thready and of low tension; his temperature was slightly subnormal, and his pale lips, and ashen pinched countenance showed well the degree of shock in which he was. His pain was severe enough to make him groan constantly, and successful catheterization of the distended bladder gave him no relief from his suffering. His abdominal wall showed no external evidence of injury; it was distended, and everywhere rigid, the muscular spasm and tenderness were general. Dulness was absent even in the flanks; the percussion note was tympanitic everywhere. On his back, over the tenth rib of the left side, and about three inches from the middle line was a red contused area, somewhat larger in size than a silver quarter. This area was tender and indicated the point of impact of the shaft. The urine was negative.

Immediate operation was advised, but the patient refused. A high enema containing turpentine brought away much gas, but little fæcal matter. A quarter of a grain of morphine subcutaneously gave him only slight relief.

Eighteen hours after the accident, having grown steadily worse, the patient consented to operation. He then showed all signs of a diffuse, general peritonitis, except vomiting. His pulse had grown weaker, and had risen to 112.

Under ether a three-inch incision was made in the median line below the umbilicus. Later it became necessary to extend this about two inches above the umbilicus. Upon opening the peritoneum considerable dark brown fluid escaped, some of which was evidently bile-stained. It brought with it many flakes of fibrin and particles of partly digested food. The small intestine presented an angry looking, congested peritoneal coat, and a general very free formation of fibrinous exudate. After a rather protracted search, a perforation which would easily admit the top of the forefinger was found in the jejunum, opposite to its mesenteric attachment, and about two and one-half inches from its beginning; through it there was a very free escape of the intestinal contents. Extending downward from it in the long axis of the intestine was a tear through the peritoneal coat of the bowel, one and one-half inches long. The closure of the perforation, which was accomplished by a purse-string suture of silk, was a matter of considerable difficulty; for the jejunum at this point could not be brought outside the peritoneal cavity, and the abdominal wall, even under ether, remained very rigid. The longitudinal tear was repaired by a single row of the Cushing right-angle suture.

At about the mid-point of the ileum for a distance of six inches both the intestine and its mesentery were much thickened; the vessels of the mesentery looked as if thrombosed. However, the circulation of the intestine appeared to be all right, and the loop was replaced. Examination of the kidneys, spleen, stomach, liver, and gall-bladder gave no evidence of injury. Several ounces of a solution of Epsom salts were injected into the cavity of the small intestine. The peritoneal cavity was irrigated with hot salt solution, and the abdominal wound hastily closed except at the lower end, where sufficient space was left for the passage of two glass drainage tubes into the pelvis. The patient failed rapidly during the operation despite stimulation. The head of his bed was raised and he was given salt solution intravenously. He reacted rapidly and well from the shock of the operation. No vomiting followed, his pain ceased, and by evening his pulse was of much better quality than it had been at any time since his entrance to the hospital.

About six hours after the operation his pelvis was irrigated with hot salt solution through the glass drainage tubes, and much cloudy fluid, with many pieces of fibrinous exudate, was washed out. Rectal feeding was started.

The morning following the operation the patient was given an enema and had a large fæcal movement. Another intravenous infusion of salt solution was given and small quantities of water were allowed by mouth. About mid-afternoon he had a slight attack of vomiting. His pulse was steady and of good volume. Late in the afternoon his pelvis was again washed out through the drainage tubes, and at midnight he was once more infused with salt solution.

He continued to improve till the afternoon of the second day, when a severe diarrhœa set in; this was controlled by opium, but his pulse began to rise rapidly. He reacted once more after another infusion of salt solution, and on the morning of the third day his pulse was steady and about 84. During the afternoon of the third day he went to pieces again and never rallied, dying about seventy-two hours after the operation.

Case 6. — Dr. Bottomley.

December 8, 1901, a boy of eight was brought to the City Hospital with a history of having been run over by a team about five hours before. There had been no loss of consciousness at the time of the accident. The boy had complained of considerable pain about the umbilicus and had vomited four or five times before coming to the hospital. He had passed perfectly clear urine.

The patient showed moderate shock. His pulse was 108, his temperature, 101; he was pale and somewhat restless. His abdomen was not distended and the percussion note was dull everywhere. There was general tenderness and muscular spasm of the abdominal wall, both being most marked over the right side of the abdomen; the spasm, however, seemed to be mostly voluntary and later under ether proved to be so. An area of ecchymosis with superficial abrasion occupied the space between the costal margin and the crest of the ileum on the right side.

Under ether an incision was made in the middle line below the umbilicus. When the peritoneal cavity was opened a large quantity of cloudy, blood-tinged serum escaped. After a short search a perforation of the intestine was found at about the mid-point of the ileum, one inch from its attachment to the mesentery and one-third of an inch in diameter; the mucous membrane was pouting through the everted edges of the opening and had prevented the escape of much faecal matter. A double row of Lembert silk sutures closed the opening. The cavity was thoroughly irrigated with hot salt solution and both flanks and pelvis were drained by tubes and wicks. The external wound was partially closed by through and through sutures of silkworm gut.

The boy's convalescence was steady and uneventful.

Case 5 is unique. Neither from the patient's story nor from that of witnesses, nor from any external sign, could we get evidence of any direct contusion of the anterior abdominal wall. He was knocked down on a wharf, on a fairly even surface, presenting no projections against which he could have been thrown. It seems therefore most probable that in this case the rupture was from within, and was the result of sort of a contre-coup (if the term may be used here). It is well known that perforation of the intestine in traumatic cases has occurred from within the bowel, the fluid contents of a loop of gut being confined for the time in a narrow space and by the impact of the violence, being thrown into a state of tension sufficiently high to burst through the intestinal wall. Makins of London mentions such cases.

Several cases are on record which had been caused by falls on the back. In all of these the resulting perforation had

taken place very close to the beginning of the jejunum, where the gut emerges from beneath and is held down by a fold of peritoneum. The intestine at this point has but little mobility, and the blow on the back would give the intestine a sudden, powerful impulse on account of the rupture. In this case the longitudinal tear in the *external* coat of the intestine adds greatly to the probability of its being caused from within.

The important feature, the one thing that means most to both patient and surgeon in all these cases, is the length of time allowed to elapse between the hour of the accident and the hour of operation. Beyond the fourth or fifth hour every hour of delay adds greatly to the danger of a fatal issue. We cannot wait for signs that indicate certain perforation of the intestine. None of us would hesitate to do an immediate operation upon a patient who we *knew* had a perforation ; we would consider an operation his only chance for recovery. But in the great majority of cases we cannot know except by operation, and the best time to know is to know early when the knowledge will be of service to the patient.

VII.

GENERAL PURULENT PERITONITIS, WITH A REPORT OF THIRTY-FIVE CASES, OF WHICH FIFTEEN RECOVERED.

By Edwin Welles Dwight, M.D., Assistant Visiting Surgeon to the Boston City Hospital.

That no one method of treatment is entirely satisfactory in the care of cases of general purulent peritonitis is apparent from the great variety that are recommended and employed. The condition is a common one, and one upon which the gravest prognosis is usually based. When a well-developed, purulent peritonitis is established, the strength of the patient and the rapidity of its onset have apparently little to do with the ultimate result. Improved methods of treatment and early operation have tended to diminish to a very large extent the frequency of this condition; when, however, it exists, the treatment now employed is not far advanced over that of twenty years ago. We appreciate the necessity of anticipating it, as we do the necessity for early operation when it appears, but after it is once well established the prognosis differs very little from that in the earlier history of abdominal surgery.

In connection with the treatment it is necessary to take into consideration the source of infection, its extent and character, and the condition of the patient affected. Death from general peritonitis is the result of toxæmia, poisons being absorbed from two general sources, the peritoneal cavity and the intestines, the rapidity of the death depending upon the resistance of the patient and the character and dose of the poison. The treatment, therefore, is directed toward

the removal of the source, the diminution of the dose, and the increase of resisting power on the part of the patient. These facts are well known, well recognized, and upon them are based almost, if not quite, all the great variety of treatments that have been suggested.

The methods that I have employed in the following cases are not unique or remarkable, their intent has been to diminish the dose with the least possible interference with the strength and viability of the patient. It is a well-recognized fact, no matter what the strength of the patient is, that the constantly increasing toxæmia in a well-developed, purulent peritonitis will be sufficient to cause death in a short time. While it is apparently a fact that certain well-developed cases of general peritonitis have recovered without operation, their number is so small, and the possibility of error so prominent, it is hardly necessary to consider them. The absorption of toxic materials from the intestines may be greatly diminished, if not entirely stopped, by a thorough and persistent emptying of the bowel; it is, however, probably impossible to control the development of bacteria and their toxic products in the peritoneal cavity. In order that the bacteria may be destroyed and their products removed, it is necessary that an opening should be made into that cavity, that in so far as possible the septic matter should be removed, and that a condition should be left behind that is not favorable to the development of these organisms.

In carrying out this idea extreme measures have been urged. The bowels have been entirely removed from the abdominal cavity, thoroughly wiped off with dry sponges, every minute part being gone over in an attempt to remove all septic products and mechanically interfere with the growth of bacteria. Patients have been placed in a bath-tub filled with mild antiseptics after the abdominal cavity has been opened, in an attempt to thoroughly cleanse the peritoneum by constant washing. Some surgeons have recommended the free use of opiates, in order that paralysis of peristalsis should diminish the transfer of septic matter from one portion of the peritoneum to another. Others of equal skill and experience strongly urge the free use of cathartics

to remove toxic materials from the intestines and assist the peritoneum in the absorption and destruction of bacteria and their products. None of these methods are entirely satisfactory, and it would seem that their want of success has been at least partially due to the fact that proper consideration has not been given to the effect upon the resistance of the patient of prolonged operations, free catharsis, or the use of powerful drugs.

It is well recognized, both experimentally and clinically, that the normal peritoneum is able to take care of very considerable doses, both of bacteria and their products, provided the resistance of the patient is good and there is no mechanical source of irritation to hamper the peritoneum in its action. Well-known experiments in the way of injecting into the normal peritoneal cavity varying doses of bacteria, alone and in connection with sterile water and sterile glass, are familiar to us all, but they have, I think, a more important bearing upon the treatment of general peritonitis than is ordinarily accorded them. The presence of a colony of bacteria within the abdominal cavity is of little moment if the conditions for their growth are not good, if there is no further source of irritation, and the resistance of the patient can be maintained.

The treatment followed in the cases that are reported in this paper is based upon an attempt to diminish the doseage to the greatest possible extent, with the least possible interference with the resistance of the patient, and from the time the case is first seen until recovery or death takes place these two objects should be kept constantly in mind, it being appreciated that if the vitality is lowered beyond a reasonable point the diminution of the doseage is of little moment. An operation, the effect of which so enfeebles the patient as to cause his death upon the operating table, or leaves him with practically no resisting power, is worse than useless, no matter how thoroughly the toilet of the peritoneum has been performed.

It will be assumed that the necessity for immediate operation is recognized when the diagnosis of general peritonitis is made. If the patient shows signs of septic absorption,

everything else should be sacrificed to the two points of diminishing the dose and maintaining the resistance. For this reason, all extreme attempts at asepsis of the skin and delays in preparation should be avoided, the patient placed upon the operating table with the least possible exposure and exertion, and lightly etherized, never going beyond the stage of stimulation. It is perfectly possible to shave, satisfactorily cleanse, operate and complete the dressing without going beyond a stage of anæsthesia which allows of talking and more or less muscular movement on the part of the patient. Stimulants are rarely given before the operation, as when ether is used in this way it acts as a strong stimulant, and is the only one that is necessary at this stage.

The abdomen is shaved, cleansed with soap and water, followed by some antiseptic, great care being taken that an excess of fluid is not used, which only serves to wet the clothing and the patient. An incision is made over the source of infection, if this be known; if not, the incision should be made in the median line, either over the pubes or half way between the ensiform and the umbilicus, according to the probability that the source of the trouble is in the lower or upper half of the abdomen. This incision should be only as long as is necessary to admit of moderate exploration and for the introduction of the tubes used in flushing the abdominal cavity.

A one-and-a-half or two-and-a-half inch incision is long enough for this purpose. A short incision has many advantages, not the least of which is that when the fingers or a tube are introduced through it the opening is sufficiently closed to keep the coils of intestine from protruding. If purulent fluid is found free in the abdominal cavity prolonged exploration is contra-indicated. If the offending part, as an appendix or Fallopian tube, presents itself, it should be tied off as rapidly as possible. If it is not easily found, no extended effort should be made to discover it. Through this opening a large glass flushing tube, about one inch in diameter and twelve inches long, is introduced, and through it is poured a large quantity of normal salt solution, which should be as hot as it can be borne with comfort on the back

of the hand. It is perfectly possible to carry the end of this long flushing tube to every part of the peritoneal cavity, and after the abdomen is thoroughly distended with the salt solution it is easy to allow its discharge through the opening at the side of the tube. This flushing should be continued until the fluid returns from all portions of the peritoneal cavity entirely clear. When it does this, the tube is removed, the excess of fluid allowed to escape, and three or four gauze wicks introduced in various directions to establish drainage. If ether has been given properly, and the patient is not *in extremis*, it is very rare that there is not time for this complete washing, and, the ether acting as a stimulant, it is usual that the pulse is better at the end of the operation than at its beginning. The quantity of salt solution used is frequently very large; from twenty to twenty-five two-quart bottles have often been used before the fluid returned in a satisfactory condition. If the solution is sufficiently hot, the effect of such prolonged washing is stimulating rather than depressing. Stimulants are of course used if at any time they are strongly indicated, but if the time arrives when stimulants are indicated the operation should cease at once, as when carried beyond this point more harm than good is accomplished.

If this method is followed out accurately and consistently, the toxic doseage is reduced to a minimum, the least possible traumatism applied to the peritoneum, and the anæsthetic and hot salt solution act as stimulants rather than tend to reduce the resistance of the patient. Strict asepsis in preparation of the patient, and possibly in operating, may be impossible, but if the patient lives long enough to develop septic processes in the skin, the chances are largely in favor of recovery, and when under constant observation and frequently dressed, as such cases should be, I have never seen any ill effects arise from a lack of preparation.

The after treatment is comparatively simple. Nourishment should be given within twelve hours in as large quantities as can be borne. If vomiting persists, feeding should be by the rectum. Stimulants should be used in moderation, the patient's strength built up by nourishing food as rapidly

as possible. If, as frequently happens, small, localized abscesses develop in the recesses of the peritoneum, primary ether is again given, the abscess cavity emptied, and the treatment continued as before.

The cases that are reported in this paper are taken in the order in which they occurred, none being included in which there was not a real purulent peritonitis. Cases in which were found considerable amounts of serum or fibrinous exudate are not included; neither are those, such as ruptures of the intestines or stomach, where the peritoneal condition was sufficiently foul, but pus, or a distinctly purulent effusion, had not developed. In the cases where the duration was short, as from twenty-four to forty-eight hours, the fluid in the peritoneal cavity was found distinctly purulent in character.

There seems to be no set of symptoms upon whose presence or absence a reasonably definite prognosis can be based. Several of the cases that recovered were in the most profound collapse and showed all the symptoms of extreme toxæmia. Several of those that died were in a fair condition at the beginning of the operation, but lost strength rapidly within a few hours. The relative height of temperature and character of the pulse is of little service in this connection, and the age of the patient is of equally small moment.

The source of infection, that is, whether from the appendix, Fallopian tubes, or elsewhere, had apparently little to do with the prognosis. Among these few cases the percentage of recovery was somewhat in favor of appendicitis. This proportion, however, was not sufficiently marked to be of any special value. On account of the character of the disease, and the condition in which most of these patients were at the time of operation, cultures were not regularly taken from the peritoneal cavity, so that the facts are wanting to prove whether or not special varieties of infection had an important bearing upon the mortality. The impression gained, however, was that this is one, if not the most important element which controlled the prognosis. Cases of induced abortion, where one of the organisms present was the streptococcus or staphylococcus aureus, rarely recovered.

Another factor which might well be considered of great importance in controlling the prognosis, is that of the time that elapsed after the infection began; but this is not borne out by the figures in these few reported cases, among which the average duration in the cases that died was sixty-nine hours, while that in those that recovered was seventy-seven hours.

While a mortality of 57 per cent. is not considered at all satisfactory, it is believed that this proportion is a very much better one than usually follows a true general purulent peritonitis, and it is believed that a very much better result will follow provided the principles controlling this method of treatment are even more thoroughly appreciated in the future than they have been in the past.

The main difficulty that I have found has been that of controlling a desire to continue the washing beyond a point where the condition of the patient would justify it, and I believe that in many of the cases herein reported I have made the mistake of doing too much.

CASE I.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Male. 21. Good.	Gangrenous Appendicitis.	Four days' duration. Acute pain; vomiting; chills.	Temp. 101.6. Pulse 120. Rigid abdomen. Fair strength.	2½-inch incision McBurney's point, followed by discharge of purulent, foul-smelling fluid from general cavity. Peritoneum generally injected. Gangrenous appendix removed.	Fecal fistula. Gradual improvement for 18 days, then vomiting, constipation, irregular temperature, and death on 20th day.	20 days.	Death.	No autopsy.

CASE II.

Male. 14. Good.	Appendicitis.	Four days' duration. Vomiting; acute abdominal pain; obstipation.	Temp. 99. Pulse 140. Extremities cold; abdominal rigidity and tenderness; tympanites.	Primary anæsthesia. 1½-inch incision McBurney's point. Abdominal cavity bathed in thin, foul-smelling pus. Irrigation salt solution. Tube drainage.	Not materially changed after operation. Became delirious, and died 12 hours after admission.	12 hours.	Death.	No autopsy.

CASE III.

Male. 44. Several indefinite attacks.	Appendicitis.	Three days' duration. Acute pain; subnormal temperature; chills, vomiting, and delirium.	"In state of collapse." Extremities cold; pulse imperceptible; abdomen tense.	Primary anæsthesia. 2-inch incision median line. Large amount of thin, turbid pus, with fibrin. Peritoneum congested. Irrigation salt solution. Glass drainage.	No marked change. Temperature 103.6. Failed steadily.	12 hours.	Death.	No autopsy.

CASE IV.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Female. 21. Good.	Salpingitis.	Two months' duration. Left pelvic pain; vomiting. In bed two weeks. Continued dull pain, but able to do a little work. 24 hours before entrance acute increase in pain; vomiting.	Temp. 101. Pulse 120. Abdomen distended, tympanitic. General rigidity and tenderness.	Incision median line. General abdominal cavity bathed in pus. Local abscess about left tube. Irrigation salt solution. Gauze drainage.	Rapid improvement. No pain or vomiting after operation. Uneventful recovery. Discharged, healed, 35 days after operation.	35 days.	Recovery.	Returned 3 months later for removal of tubes.

CASE V.

Female. 24. Good until two months previous.	Salpingitis.	Two months previous, sudden pain right iliac. In bed 4 weeks, vomiting, etc. 2 weeks previous, uterus curetted. Constant pain right side. Vomiting 6 hours before entrance, chill and increase in pain.	Temp. 105. Pulse 145. Abdomen distended, tympanitic. Rapid collapse.	Primary anæsthesia. 4-inch incision median line. Large amount of thin, gray pus from general cavity. Tubes not removed. Irrigation salt solution. Glass drainage.	Temporary improvement for 48 hours; then failure. Abdominal cavity irrigated salt solution. No improvement.	3 days.	Death.	No autopsy.

CASE VI.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Female. 36. Salp. 9 yrs.	Salpingitis.	Forty-eight hours' duration. Chills, pain and vomiting.	Temp. 102.8. Pulse 120, irregular. Face flushed and anxious. No distension or rigidity.	Three-inch incision median line. General purulent peritonitis. Large abscess about uterus, with adhesions. Right tube removed. Irrigation salt solution. Gauze drainage.	Some hiccough. Persistent pain for 3 days. Temp. up for 8 days, then normal. Uneventful convalescence. Discharged healed.	47 days.	Recovery.	

CASE VII.

Male. 46. Abdominal pain. 1 year.		Forty-eight hours' duration. Acute abdominal pain; vomiting.	Cachetic, cyanotic. Temp. 102. Pulse 120. Abdomen distended, tympanitic, tender.	Three incisions. Large amount yellow pus in general cavity. No cause found. Irrigation salt solution. Gauze drainage.	Temporary improvement; then rapid failure.	24 hours.	Death.	No autopsy.

CASE VIII.

Female. Unknown. Good.	Salpingitis.	Five days' duration. Severe pains, chills and vomiting.	Temp. 103.6. Pulse 160. Intense stercoraceous vomiting. Abdomen distended, tympanitic, rigid.	Primary anæsthesia; 2-inch incision median line. Adhesions in pelvis. General cavity filled with grayish, purulent material. Irrigation salt solution. Glass drainage.	Temporary improvement; then collapse.	18 hours.	Death.	No autopsy. Streptococcus infection.

CASE IX.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Female. 18. Indefinite.	Unknown.	Thirty-six hours' duration. Pains; chills; collapse.	Collapse. cyanotic. Pulse almost imperceptible. Abdomen distended, tympanitic, rigid and tender.	Primary anæsthesia; 2-inch incision median line. Large amount yellow sero-pus. Irrigation salt solution. Gauze drainage.	Temporary improvement; then rapid collapse.	18 hours.	Death.	Pneumococcus.

CASE X.

Female. Unknown. Negative.	Salpingitis.	Over exertion two weeks previous, followed by pain; 48 hours previous acute pain till 6 hrs. before entrance. Vomiting.	Collapse; respiration rapid and shallow. Temp. 102. Pulse abt. 200. Abdomen distended, tympanitic, congested, and tender.	Primary anæsthesia; 2-inch incision median line. Yellow pus and flakes in general cavity. Abscess in pelvis. Irrigation salt solution. Tube drainage.	Temporary improvement, then rapid collapse.	10 hours.	Death.	No autopsy.

CASE XI.

Male. 29. One previous attack.	Appendicitis.	Two days' duration. Pain; vomiting, with a little blood.	Fever; obstipation; abdomen distended, tender, rigid, and tympanitic.	Primary anæsthesia; 2-inch incision McBurney's point. Purulent fluid in general cavity. Injection of peritoneum. Some adhesions. Perforated appendix removed. Irrigation salt solution. Gauze drainage.	Temporary improvement; sudden collapse.	6 days.	Death.	No autopsy. Streptococcus.

CASE XII.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Male. 32. Negative.	Typhoid fever perforations.	Typhoid fever 14 days. Day before entrance sudden pain in right lower abdomen.	Tenderness, rigidity, and distension. Pulse rapid and fair. Temp. 99.	2½-inch incision McBurney's point. Turbid, purulent fluid in general cavity. Injection of peritoneum. Appendix much inflamed, and removed. Perforatory ulcer sutured. Irrigation salt solution. Glass drainage.	Temporary improvement; 5 days later wound dilated. Pockets with pus evacuated. Gradual failure.	7 days.	Death.	No autopsy.

CASE XIII.

Female. 28. Negative.	Salpingitis.	Acute pain 6 days. Chills; nausea.	Temp. 101. Pulse 120. Distension, rigidity and tenderness.	Incision median line. Turbid, purulent fluid in general cavity. Peritoneum injected. Abscess about left tube. Irrigation salt solution. Tube drainage.	Temporary improvement. All symptoms diminishing. Apparently doing perfectly well. Sudden pain and collapse.	21 days.	Death.	Autopsy showed double pyosalpynx. Abdomen clean. No cause for death.

CASE XIV.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Male. 21. Negative.	Appendicitis.	For several days sharp, diffuse abdominal pain. Vomiting.	Temp. normal. Pulse 120. Fair strength. No distension; general tenderness.	Two-inch incision McBurney's point. Thin, foul puriform fluid in abdominal cavity. Peritoneum injected. No adhesions. Gangrenous appendix and omentum removed. Irrigation salt solution. Tube drainage.	First three days delirious. Pulse 140. Fecal discharge, then improvement and uneventful convalescence.	52 days.	Recovery.	Entirely healed.

CASE XV.

Female. 20. Negative.	Salpingitis.	For six days chills, acute pain, vomiting.	Temp. 101. Pulse 140. Distension, tympanites and tenderness.	Four-inch incision median line. Purulent fluid in general cavity. Many fine adhesions. Both tubes removed.	Rapid improvement. Uneventful convalescence.	45 days.	Recovery.	Gonnococcus and pneumococcus.

CASE XVI.

Female. Age unknown Not asked.	Induced abortion.		Pulse 180. General tenderness; distension. No rigidity.	Median incision. Fecal pus in general cavity. Both tubes and uterus injected and soft. Irrigation salt solution. Gause drainage.	Temporary improvement, then delirium and rapid failure.	4 days.	Death.	No autopsy.

CASE XVII.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Female. 30. Negative.	Salpingitis.	Five days' acute, general abdominal pain. Vomiting; chills.	Distension, rigidity, and general tenderness.	Median incision. Double salpingitis. Tubes not removed on account of weak condition. Irrigation. Gauze drainage.	Rapid improvement. Uneventful convalescence.	37 days.	Recovery.	

CASE XVIII.

Male. 22. 8 days ago inflammation of the bowels.	Appendicitis.	Three days' duration. Sudden, acute abdominal pain. Vomiting.	Temp. 102. Pulse 120. General rigidity and tenderness.	Primary anæsthesia. Incision McBurney's point. Purulent fluid in general cavity. Inflammation of peritoneum. Appendix not removed on account of general condition of patient. Irrigation salt solution. Gauze drainage.	No marked improvement. Gradual failure.	4 days.	Death.	No autopsy.

CASE XIX.

Female. Unknown. Negative.	Miscarriage.	Negative.	Temp. 103. Pulse 160. Distension, rigidity and general tenderness.	Median incision. Purulent fluid in general cavity. Fine adhesions. Injection of peritoneum. Tubes not removed on account of general condition. Irrigation salt solution. Gauze drainage.	Marked temporary improvement; then rapid failure.	5 days.	Death.	No autopsy.

CASE XX.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Female. 18. Two previous attacks.	Appendicitis.	Twenty-four hours' duration. Acute pain; chills; vomiting; constipation.	Temp. 99. Pulse 120. Fair strength. Tenderness, rigidity and distension.	Two-inch incision McBurney's point. Purulent fluid in general cavity. Gangrenous appendix removed. Irrigation salt solution. Gauze drainage.	Gradual improvement for one week. Uneventful convalescence.	45 days.	Recovery.	

CASE XXI.

Male. 48. Five previous attacks.	Appendicitis.	Two days' duration. Acute pain; vomiting; chills.	Temp. 100. Pulse 78. Moderate distension; rigidity; diffuse tenderness.	Three-inch incision McBurney's point. Free, purulent fluid in general cavity. Peritoneum injected. Some fine adhesions. Gangrenous appendix removed. Irrigation salt solution. Gauze drainage.	Fecal fistula. Gradual improvement for 10 days, then uneventful convalescence.	51 days.	Recovery.	

CASE XXII.

Male. 21. Negative.	Appendicitis.	Twenty-four hours' duration. Acute pain; chills; vomiting.	Temp. 102.6. Pulse 120.	Two-inch incision McBurney's point. Purulent fluid in general cavity. Gangrenous appendix removed. Irrigation salt solution. Gauze drainage.	Fecal fistula. Gradual improvement for 10 days, then uneventful convalescence.	36 days.	Recovery.	

CASE XXIII.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Female. 15. Negative.	Appendicitis.	Seven days' duration. Constant pain; constipation; fecal vomiting; chills.	Temp. 100. Pulse 90. Distension, rigidity and tenderness.	Two-inch incision McBurney's point. Puriform fluid in general cavity. Local abscess about appendix. Perforated appendix removed. Irrigation salt solution. Gauze drainage.	Rapid improvement. Uneventful convalescence.	32 days.	Recovery.	

CASE XXIV.

Male. 42. Negative.	Abscess of liver.	One week's duration. Acute pain; chills; vomiting, Temporary improvement, then recurrence of symptoms 24 hours before entrance.	Temp. 100.2 Pulse 80. Rigidity, distension and tenderness.	Incision over appendix. Purulent fluid in general cavity. Slightly inflamed appendix removed; also lower ensiform. Adhesions. Abscess. Irrigation salt solution. Gauze drainage.	Rapid improvement for 12 days, then pocketing of pus. Drained. Uneventful convalescence.	27 days.	Recovery.	

CASE XXV.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Female. 15. Negative.	Appendicitis.	Four days' duration. Pain; chills; vomiting.	Temp. 102. Pulse 120. Condition poor. Distension; tympanites; rigidity; tenderness.	Two-inch incision McBurney's point. Purulent fluid with fecal odor in general cavity. Diffuse, light adhesions. Gangrenous, perforated appendix removed. Irrigation salt solution. Gauze drainage.	Rapid improvement for one week, then chill; pocketing of pus. Drained. Three days later purulent discharge from vagina; four days later parotid abscess. Pocketing of pus on 30th day. Uneventful convalescence.	51 days.	Recovery.	Staphylococcus pyogenis. aureus.

CASE XXVI.

Male. 30. Negative.	Appendicitis.	Three days' duration. Acute abdominal pain; chill; nausea; vomiting; constipation.	Temp. 97. Pulse 95, weak and irregular.	2½-inch incision McBurney's point. Purulent fluid in general cavity. Peritoneum injected. Gangrenous appendix removed. Irrigation salt solution. Gauze drainage.	No marked improvement. Increasing temperature; failure. Death in four days.	5 days.	Death.	No autopsy.

CASE XXVII.

Female. 43. Negative.	Salpingitis.	Eight days' duration. Pain in back and abdomen. Fever; vomiting; obstipation.	Temp. 103. Pulse 140, weak and intermittent. General distension, resistance, and tenderness.	Primary anæsthesia; 3-inch incision McBurney's point. Pus in general cavity. Appendix normal. Adhesions in pelvis. Irrigation salt solution. Gauze drainage.	Patient was in extremely critical condition at time of operation. Rallied for a few hours; gradually failed, and died eighteen hours after operation.	20 hours.	Death.	No autopsy.

CASE XXVIII.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Male. 27. Negative.	Appendicitis.	Thirty-six hours' duration. Acute abdominal pain; vomiting; no chill.	Temp. 103. Pulse 100. Slight distension: tympanites; rigidity; general tenderness.	Primary anæsthesia. Incision McBurney's point. Pus in general cavity. Gangrenous appendix removed. Irrigation salt solution. Gauze drainage.	Temporary improvement for 3 days, then failure.	6 days.	Death.	No. autopsy.

CASE XXIX.

Male. 56. Gastric disturbance several years.	Perforating ulcer of the stomach.	Gastric symptoms 3 weeks. In bed 36 hours. Acute pain; vomiting small amounts; chills.	Temp. 101. Pulse 80. Poor. Dulness in left chest. Distension; tympanites; rigidity; tenderness.	Primary anæsthesia. 2-inch incision McBurney's point. 3-inch incision, median line, below ensiform. Purulent ulcer in stomach, with abscess, which had broken into general cavity. Ulceration closed. Irrigation salt solution. Gauze drainage.	No marked improvement. Vomiting continued. Temp. below 100.	4 days.	Death.	Autopsy. Non-malignant ulcer. Bronchopneumonia.

CASE XXX.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Female. 37. Many similar attacks.	Appendicitis. Salpingitis. Perforation of colon.	Eleven days' acute abdominal pain; vomiting.	Temp. 103. Pulse 130, irregular. Rigidity; distension; tenderness.	Five-inch incision median line. Pus and fæces in general cavity. Adhesions in pelvis and about appendix. Perforation of cæcum about 1 cm. in diameter. Large fibroid and one pus tube. Perforated appendix removed and cæcal opening closed. Irrigation salt solution. Gauze drainage.	Temporary improvement for 36 hours, then rapid failure and death in 36 hours after operation.	6 days.	Death.	No autopsy.

CASE XXXI.

Male. 29. Negative.	Appendicitis.	Three days' acute pain; chills; vomiting.	Temp. 103.5. Pulse 120. Distension, rigidity and tenderness.	2½-inch incision McBurney's point. Puriform fluid in general cavity. Peritoneum injected. Gangrenous appendix removed. Few light adhesions. Irrigation salt solution. Gauze drainage.	Improvement for 15 days, then pocketing of pus. Ether; drainage established. Uneventful convalescence.	39 days.	Recovery.	

CASE XXXII.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Male. 19. Negative.	Appendicitis.	Two days' acute pain; vomiting; obstipation.	Temp. 103.6. Pulse 120.	Two-inch incision McBurney's point. Thin, yellow pus in general cavity. Appendix not removed on account of adhesions and condition of patient. Irrigation salt solution. Gauze drainage.	Temporary improvement for 3 days, then pocketing of pus. Drained. Gradual failure.	6 days.	Death.	No autopsy.

CASE XXXIII.

Male. 30.	Gangrenous appendicitis.	Eight days' acute pain; vomiting; chills.	Temp. high. Pulse 160. Peritoneum injected. Profound sepsis. Very weak.	Primary anæsthesia. 3-inch incision McBurney's point. Free pus in general cavity. Peritoneum injected. Irrigation salt solution. Gauze drainage.	Fecal fistula. Slow, uneventful convalescence.	2 mos.	Recovery.	Persistent sinus. Second operation one year later. Recovery.

CASE XXXIV.

Female. 28. Symptoms two years.	Salpingitis.	Three days' acute pain; vomiting; chills.	Temp. 101. Pulse 120. Abdomen distended, rigid, tympanitic.	Three-inch incision median line. General cavity filled with purulent material. Local abscess of right tube. Tube removed. Irrigagation salt solution. Gauze drainage.	Uneventful convalescence.	21 days.	Recovery.	Second operation for adhesions one year later. Recovery.

CASE XXXV.

Sex, Age, and Previous History.	Cause.	Present Illness.	Condition.	Operation.	Condition in Hospital.	Time in Hospital.	Result.	Remarks.
Female. 18. Several previous attacks.	Gangrenous appendicitis.	Eight days' acute pain; vomiting; chills.	Temp. 103. Pulse rapid. Fair strength. Abdomen distended, tympanitic. General rigidity.	Two-inch incision McBurney's point. Sloughing appendix removed. Peritoneal cavity filled with foul-smelling pus. Local adhesions about appendix. Irrigation salt solution. Gauze drainage.	Pocketing of pus at 2 weeks. Drained under ether. Uneventful convalescence.	2 Mos.	Recovery.	

VIII.

SURGICAL TREATMENT OF HEMORRHAGIC PACHYMENINGITIS.

By John C. Munro, M.D.

That cerebral pachymeningitis is not rare is demonstrated by the results of autopsies, which show this lesion in from 1 to 10 per cent. of all cases, a large proportion occurring in the insane, and a small proportion in infants. Neither of these types will be considered here. It is to the lesion occurring in adults just beyond the prime of life, not insane, often stricken down in the street to which I wish to call attention.

The pathology and definite etiology of this disease is still a matter of discussion, and is beyond the province of this paper. In fact, the interest heretofore has been almost exclusively pathological; that it should be more frequently surgical I wish to emphasize.

In three of the eleven cases reported the diagnosis is a clinical one, and confirmed, so far as may be, by operation. Absolute pathological diagnosis is lacking, and yet it has seemed best to include these cases. In entering upon a comparatively new surgical field like this, it is inevitable that certain cases should be accepted on insufficient data, to be eliminated later perchance when our clinical knowledge becomes more exact.

Bullard, in a paper read before the American Neurological Association in 1895, reporting a case in which he diagnosticated and advised operation for hemorrhagic pachymeningitis, says "The great importance of diagnosis in these cases of subdural hemorrhages lies in the fact that many of them are probably susceptible to proper treatment, and their im-

mediate fatal effects can be prevented. Probably we cannot as yet prevent the recurrence of new hemorrhages later." His experience, and my own, since that was written, seven years ago, bears out the importance of bringing up this subject again.

In analyzing the clinical histories of my cases, one must bear in mind that much of the data must be obtained from the family or friends, poor data at best. Moreover, the patient may be mentally unbalanced at the onset of the disease, though otherwise not insane, being ignorant of his condition, whereabouts, etc. In fulminating cases no history can be obtained beyond that of probable injury, intoxication or assault. Other concomitant diseases, acute or chronic, may so mask the cerebral condition that very little regard is paid to the latter, and its true significance is not appreciated. Granted that a reasonable diagnosis of subdural hemorrhage can be made, an operation by a competent surgeon, with a view to determine the exact condition, is practically harmless. I cannot help believing that an occasional case of so-called fracture of the base, alcoholic œdema or apoplexy, is left to die, when a timely trephining and evacuation of a clot due to a pachymeningitis, would save life. In other words, barring the presence of other grave organic lesions, pachymeningitis hemorrhagica, when severe enough to produce evidences of cerebral irritation or pressure, is distinctly a surgical disease, and the expectant treatment by cathartics, ice-caps, etc., is not warranted in the light of modern surgical advance.

An exact diagnosis of this disease is difficult and at times impossible. The lesion is more apt to be found in men past middle life, in chronic alcoholism, syphilis, or other diseases causing depravity in the vascular system. It may be concomitant with acute or chronic diseases, in which cases the meningeal disturbance may be slight or overlooked. Trauma apparently plays an important role, but unfortunately it tends to obscure the symptoms due to the meningeal hemorrhage otherwise present. The pressure symptoms are apt to come on slowly and irregularly. They are preceded by definite signs of irritation, at first unilateral, later bilateral but unequal.

In other words, a prolonged stage of irritation and meningeal disturbance irregular in character, followed by paralysis and stupor, even if there is a definite trauma preceding the appearance of these signs, should suggest an intercurrent hemorrhagic pachymeningitis. Convulsions may occur, although they were not noticed in any of my cases. The late appearance of the paralysis, and the repetition or multiplicity of attacks, together with the preponderance of symptoms of irritation are regarded by Bullard and other writers as the most important diagnostic signs. "More characteristic than anything else is the existence of early stiffness and rigidity in one or more limbs."—(*Bullard.*) In some instances the hemorrhage may be apparently sudden and profuse, making it impossible to distinguish this form from other forms of apoplexy, but it is always possible that there has been an earlier, unrecognized and prolonged irritation of mild degree.

As to treatment, I believe without question that trephining is the only thing to be considered, if there is the least chance of the operation itself not proving fatal. Even if a suspected meningeal hemorrhage is not found, but an alcoholic œdema, an internal hydrocephalus, a hemorrhage from fracture or other cause, there is always a possibility of at least temporary relief by drainage of the subdural space or of the ventricles. In obscure cases it is safer to trephine both sides. The operation can be done rapidly, the shock is increased but little, and in some cases accurate localization is impossible before operation.

The prognosis is not good at best, and it is made worse by prolonged irritation and pressure of the brain. These cases are surgical from the outset. The surgeon should be the one to decide when an early operation is indicated, and having decided he should operate without delay.

In analyzing briefly the following cases, all but one being taken from the recent records of the Boston City Hospital, emphasis will be placed upon the symptoms referable to the meningeal lesions. The first four cases exhibit so little evidence of disturbance from the subdural bleeding that they can be dismissed in a few words. From a diagnostic and surgical point of view they are not important.

Case 1. — John R. (Dr. J. L. Morse's service in 1901.) Farmer, forty-eight years old; very little alcohol or tobacco; arteries rigid and tortuous. Entered for symptoms not referable to a cerebral lesion, Diarrhœa, loss of weight, vomiting, etc. Autopsy showed multiple, malignant tumors of the bones, ulcerative proctitis, colitis and hemorrhagic pachymeningitis. The right dura on the inner surface presented a delicate film under which were numerous circular, bright red areas from one-half to two millimeters in diameter. Over the left hemisphere were similar areas of hemorrhage, but much fewer in number. Gray substance normal.

Case 2. — Annie P. (Dr. Geo. B. Shattuck's service, 1900.) Thirty-seven years, alcoholic (beer) for several years. Sudden delirium two days before entrance. No cerebral symptoms excepting delirium followed by coma. Autopsy showed cirrhotic and fatty liver, parenchymatous nephritis, and œdema of the brain. The under surface of the dura showed several red areas from 2 to 4 cm. in diameter, slightly elevated. From the surface of these a delicate transparent membrane could easily be stripped, leaving a smooth reddish surface. The color semed in part due to extravasated blood. On raising the pia from the cortex there seemed to be a considerably increased amount of clear fluid, and the brain tissue on section was somewhat more moist than normal.

Case 3. — Samuel P. (Dr. F. H. Williams' service in 1899.) Seventy-five years old. Paralysis and anasthesia of legs for eight months. Absent reflexes, acute pneumonia.

Autopsy showed pneumonia, acute pyelitis, cystitis, and atrophy of the brain with compensatory œdema of the pia. Gumma in right middle fossa. Dura was much thickened, and to the eye and touch seemed like velvet. Everywhere throughout were innumerable fine meshes of minute blood vessels. Opposite the left frontal prominence there appeared to be a slight free hemorrhage beneath the dura. The meshes of the arachnoid were filled with a clear fluid.

Case 4. — Herbert J. (Dr. Bolles' service in 1901.) Thirty-four years old. Entered for fracture of the base after a fall of twenty-five feet. Delirious, active and irritable, with left facial paralysis, and quick nystagmus. Reaction of extremities normal. The irritability grew less, though never disappearing, the nystagmus ceased, and four weeks after admission he became stupid and comatose, and died. His temperature up to the last few days was normal or subnormal, his pulse being low, as a rule, also.

Autopsy showed the dura to be smooth and pale, except over the base in the posterior fossa, where it was overlaid with a thin sheet of dark red hemorrhagic membrane, in some places adherent, and (2) over the right middle and anterior fossa, which showed a few sharply limited oval areas, one to two centimeters in diameter, dark red to purple in color. There were multiple areas of softening in right hemisphere. Brain substance without œdema. Fracture of the left base..

In the two following cases no operation was attempted. It is probable that it might have proven beneficial if done early enough.

Case 5. — Christopher F. (Dr. C. F. Folsom's service in 1897.) Irish laborer, forty to fifty years old, brought to hospital with history of hav-

ing been ill for two weeks, nearly unconscious at entrance, but susceptible to pain. Respiration labored, cyanosis, right eye turned out, pupils moderate size. The left pupil was contracted, neither pupil reacted, and no facial paralysis. Rigidity almost constant in left lower extremity. Occasional rigidity in right. Left upper extremity in constant motion, with scratching over left iliac region and thigh. Right upper extremity somewhat rigid, and with very little motion. Left knee-jerk not obtained on account of rigidity. Right markedly increased. No ankle clonus. Planter reflex present on both sides. No cremasteric, abdominal or epigastric reflex. Leucocytosis 19,000, positive Widal reaction. Lumbar puncture revealed considerable slightly turbid sterile fluid. Temperature 103° and increasing; pulse 105 rising to 160. Chest and abdomen negative. Death shortly after admission.

Autopsy. — On removing calvarium considerable dark brownish fluid escaped. Dura distended. On its removal, the right side was found lined with a dark brown mass of blood of 6 to 8 mm. in thickness. Convolutions depressed, but not flattened. The hemorrhage extended all under the dura of the right side and over the top of the tentorium. On the left side the dura was clean, and the convolutions were extremely flattened. At the base the optic commissure was shoved to the left, and on separating the two halves of the cerebrum the right was found projecting in the frontal region into the left half. The brain on section showed nothing remarkable. Examination of the hemorrhage showed blood mainly normal in appearance, with numerous large cells filled with blood globules. No crystals or blood pigments found.

Anatomical Diagnosis. — Chronic internal hemorrhagic pachymeningitis.

Case 6. — John W. (Dr. G. G. Sears' service in 1896.) Englishman, forty-six years old. History from wife. Always well. No alcohol. No tobacco. Four years ago an attack of hiccough. Four months ago congestion of the lungs, followed by hiccough lasting a week. Violent pain in the head followed this illness also, and has continued ever since. Five days ago hiccough began again, and was treated by his physician with chlorodyne. The day before entrance, after a single dose, he became drowsy. At admission he was unconscious; temperature, 96.4, pulse, 60, respiration, 12, and he had hiccough. He was aroused by supraorbital pressure, and the pupils, previously pinpointed, dilated and reacted to light. Patella, abdominal and plantar reflexes absent. Chest negative. Fundi and ears negative. Leucocytes 4200. With irregular attacks of hiccough and incontinence of both urine and feces he lived twelve days. At one time there was a cataleptic condition of the limbs, and towards the end pulmonary symptoms developed.

Autopsy showed the inner surface of the dura covered with a grayish membrane, which could be easily pulled off. Between this membrane and the dura was a large amount of hemorrhage, partly posteriorly, of the left side, where in places it was a centimetre in thickness. This reddish membrane extended well down into all three of the fossæ, but particularly into the middle and anterior. The convolutions were considerably depressed.

Anatomical Diagnosis. — Chronic internal hemorrhagic pachymeningitis, acute bronchitis and broncho-pneumonia.

Case 7. — Reported by Bullard, as mentioned above, from whose account I will quote freely. The patient, a man about sixty-five years of age, had used considerable alcohol for the past few years. In fairly good health, he had worked up to eight days before entrance to the Carney Hospital. Seven days before, he fell unconscious, but in two days was better, and recognized his sister. At this time there was partial paralysis of the right limbs, gradually disappearing. At entrance he was stupid, somnolent, more or less restless, and at times delirious. He moved all his limbs. There was drooping of right eyelid, and divergence of right eye. There was frequent voluntary movement of all limbs, but the left upper extremity seemed stronger than the right. Response to stimuli diminished, but more so on the right side than on the left. Knee jerks absent. On the following day he was very restless, pulling the bed-clothes and any other object he could reach with the left hand. Incontinence. Both eyes turned upwards and outwards, the left pupil being smaller than the right.

"The important symptom was the appearance of a marked right hemiplegia of the extremities, the right upper extremity being apparently totally paralysed, the right lower extremity largely so."

Sensation much diminished over right extremities. Dr. Bullard made a diagnosis of hemorrhagic pachymeningitis, and referred the case to me for immediate operation. By the time consent could be obtained the patient was nearly moribund. The left parietal eminence was trephined, and an immense clot removed from beneath the dura, extending from the anterior lobe backwards over the occipital, the thickest portion being over the vertex, and just posterior to the motor region. The false membrane was plainly visible. Pulse and respiration greatly improved at close of operation, but twenty-four hours later he again developed Cheyne-Stokes respiration and died.

Case 8. — James S. (Dr. Henry Jackson's service in 1897.) Irish laborer, forty to forty-five years old. History obtained from wife. Alcohol, but never out of work. Always strong and well. No evidence of syphilis. One week before entrance complained of malaise without definite symptoms. A day later he scuffled both his feet when he walked, and could hardly go up stairs on account of weakness and the scuffling. He then went to bed, and his speech became slow. There was considerable tremor of the right extremity, and a little of the left. Four days after he slept all day, and could not be aroused, although on the following day he was conscious, but stupid. No paralysis was noticed by his wife, but his right arm and hand were kept in constant motion, even when he was asleep, the right fingers continually picking at his clothes, as if he wished to pull open his shirt-front. This continued for a day longer until admission, when he was unconscious, voiding his urine and feces involuntarily. After admission he became partly conscious, but not rational. Slight left facial paresis, pupils equal, moderately dilated, very sluggish to light. Chest and abdomen practically negative. Knee jerks markedly diminished, plantar reflex increased. Upper right extremity in constant motion, as if tearing away his clothes. Less marked symptoms in left upper extremity. Both legs in motion most of the time.

The knee jerk and facial paresis disappeared. He grew worse mentally and physically, and after four days was seen by Dr. J. J. Thomas and myself in consultation, and transferred for operation. Trephined on the right side. Dura tense, slight œdema of the cortex, which bulged without pulsation; ventricle tapped, with escape of a large quantity of fluid, and the pulse, which was weak and 40, became of good strength and 72 to 80. Opposite side unfortunately not trephined.

Autopsy showed operation wound on right. On the left the dura was found to be discolored and œdematous. On its removal a large brownish black, rather firm clot covering the entire convexity of the left hemisphere was found, which was readily removed from the surface of the brain, but was adherent to the dura. Entire convexity of the left hemisphere was much compressed. No visible exudate beneath the pia, but a smear showed pus. No organisms could be demonstrated. Left lateral ventricle closed by pressure. On removing the cord a large amount of cloudy fluid escaped, containing an abundance of pus cells. No visible exudate.

Anatomical Diagnosis. — Left chronic hemorrhagic pachymeningitis, with œdema of the brain, slight cerebral spinal meningitis, tricuspid insufficiency, chronic passive congestion of the spleen and kidney.

The skull was opened on the wrong side, too much stress being placed on the supposed hemiplegia of the normal side. Had both sides of the skull been trephined the true condition would have been found and relieved.

Case 9. — Frank O'B. (Dr. G. G. Sears' service in 1896.) Irishman, 34 years old. History obtained from friends. Syphilis seven years ago, with treatment for three years. Not a regular hard drinker. Two months ago was unconscious for a few minutes. Two days before entrance to the hospital he complained of pain across his forehead. Went to bed feeling well, but got up during the night feeling ill with severe headache and numbness in one side. Shortly afterwards he became unconsious, with violent twitchings of his arms. At admission he was unconscious, pupils somewhat dilated, equal and reacting. Knee-jerks exaggerated, no clonus. Sense of pain normal. Could be aroused, and could answer intelligently to some extent. Recognized pin-prick, but would mistake side. Left-hand grasp weak. Could move all extremities, but when irritated invariably moved right arm. On the following day he was less easily aroused, passed urine involuntarily, the temperature gradually rose. The left pupil was slightly dilated.

Seen by Dr. Bullard, who advised operation. At operation, a few hours later, he was comatose, the pulse and temperature rising. At operation on the left side the dura was found to be blue and not pulsating. Beneath it were recent clots, covering the hemisphere from one-eighth to one-half inch in thickness, more abundant in the frontal and occipital regions. Washed clear with hot water. On the right side a small opening was made. The dura looked normal, pulsated, and was not opened. Very little shock. He never recovered conciousness after operation, but tossed about in bed and muttered unintelligently until his death, six hours later.

Case 10. — Benson, M. G., 62 years old, alcoholic. Very meagre history could be obtained, as he had not been living with his wife, and nothing was known of his recent doings. He is said to have been queer for a couple of years, but, as he had lived a dissolute life, little stress can be placed on this statement. He was picked up by the police in the street, and was able to talk when taken to the station-house. Shortly afterwards he became unconsious, and was brought to the relief station of the Boston City Hospital and trephined at once. At entrance he was totally unconscious, and could not be aroused; the right pupil was dilated, but both pupils reacted to light. No bleeding from the mouth, nose or ears. Chest and abdomen negative; temperature normal; pulse 80; right knee-jerk absent; no rigidity; hematoma in right parietal region.

Operation by Dr. Germain and myself. Trephined on the right side. no pulsation of dura, which was bulging and dark blue; beneath it was found much serum, and several ounces of blood-clot, well formed, adherent to the dura, and covering entire hemisphere, so far as could be told. It was washed out after being broken up with the finger. The brain, depressed for an inch or more, expanded partly and pulsated. Trephined also on the left side, and a diffuse, thin layer of blood-clot was found and washed out. The patient gained consciousness towards the close, and talked quite intelligently. On the day following the operation he was clear mentally at times, but delirious for the most part, making numerous attempts to get out of bed. Dressing soaked through and changed. Five days later he was clear mentally, not having been delirious for two days. Drainage removed. Ten days after operation the mental condition was good, wound healing rapidly, no disturbance of special senses. Two weeks after operation he was discharged to the Out-Patient Department.

Case 11. — Frank H. T. Cobbler, 44 years old. Brought to the Relief Station of the Boston City Hospital, by the police, September 3, 1902, unconscious, with profuse bleeding from the right ear. Transferred to my service at the main hospital on the following day. On the 5th he stated that, while intoxicated, he had fallen on the sidewalk. As it was a clear case of basal fracture no detailed history was obtained. On the day that he gave his history he was conscious but dull. The pupils were equal and reacted. No disturbance of the special senses, and no paralysis. On the 6th a right facial paralysis was noticed, knee-jerks equal, dull and delirious. He then improved gradually until the 10th, when he became unconscious, and could not be aroused. The left extremities became rigid. The facial paralysis, knee-jerks and pupils remained as before. In the afternoon he became conscious and answered questions, and the spastic condition had disappeared, but on the 11th he was rigid on both sides, more markedly so on the left, and kept picking with the left hand quite continuously. Unconsciousness returned, though not complete. Before operation the temperature ranged from 98° to 101.5°. After operation it steadily fell from 102° to normal. The pulse ranged from 100 to 60 before operation, steadily rising afterwards to 120. Dr. Bullard saw him with me in consultation, and we both independently made a diagnosis of hemorrhagic pachymeningitis. Right

side trephined. Dura tense and without pulsation. Beneath it was blood serum, and dark fluid blood coming from all over that hemisphere. Ventricle tapped, and a moderate excess of serum escaped under pressure. Pulsation returned. Then trephined on the left side, and blood serum spurted from beneath the dura, followed by dark fluid blood from all over the hemisphere. Right subdural space irrigated. No shock. He improved somewhat after operation, but died several days later.

Autopsy showed a hemorrhagic pachymeningitis on both sides, fissured fracture of right petrous bone, hemorrhagic softening in right frontal lobe, one inch in diameter, and in the left frontal lobe half-inch in diameter.

The showing in these cases is not a brilliant one so far as the results from operation is concerned. In the one case that recovered it is impossible to rule out fracture, although the evidence at the time of operation and later was against that diagnosis.

Conclusions.

1. Hemorrhagic pachymeningitis is found in the insane and in infants, but for the most part in men not insane after middle life.

2. Alcohol, syphilis, acute and wasting diseases, and trauma, apparently bear some causative relation.

3. The symptoms are those of diffuse subdural hemorrhage, coming on slowly, producing mental irritation, spasm, and rigidity of the extremities, convulsions, and later, paralysis, the sequence being more or less irregular.

4. The cranial nerves are not liable to be affected.

5. The treatment is surgical, and should be instituted as early in the disease as possible.

6. Without operative relief, in cases with pressure symptoms, the prognosis is practically hopeless. Early operation is not serious and gives the best chance for recovery.

IX.

A METHOD OF ANESTHESIA USEFUL IN OPERATIONS ON THE HEAD, FACE, MOUTH AND NECK.

By Hayward W. Cushing, M.D.

Every surgeon called upon to operate on the head or neck must have been impressed by the disadvantages of the usual methods of anesthetizing the patient during such a procedure. The usual inhaler so covers the face that during the actual inhalation of the ether operative work is impossible. Therefore the ether must be inhaled interruptedly, and consequently the anesthesia must at times be more profound than would otherwise be necessary.

Again, this interruption of the surgeon, on account of the patient's partial recovery from the anesthesia, often occurs at some critical point of his work.

This state of affairs consequently prolongs the duration of the etherization as well as impedes the operator.

Also another important factor is the difficulty of preventing infection of the operation-area from the "inhaler," or "cone," which is constantly in contact with the hands of the etherizer, and the lips, nose, saliva, mucous, or vomitus of the patient. Careful as the anesthetizer may be, it is often impossible to avoid wholly this source of danger.

On account of these unsatisfactory conditions the writer has endeavored to find some method to etherize a patient by which the etherization should be uniform and continuous, the field of operation unobstructed, and the danger of infection from the apparatus avoided.

Among the methods already on record are the following:

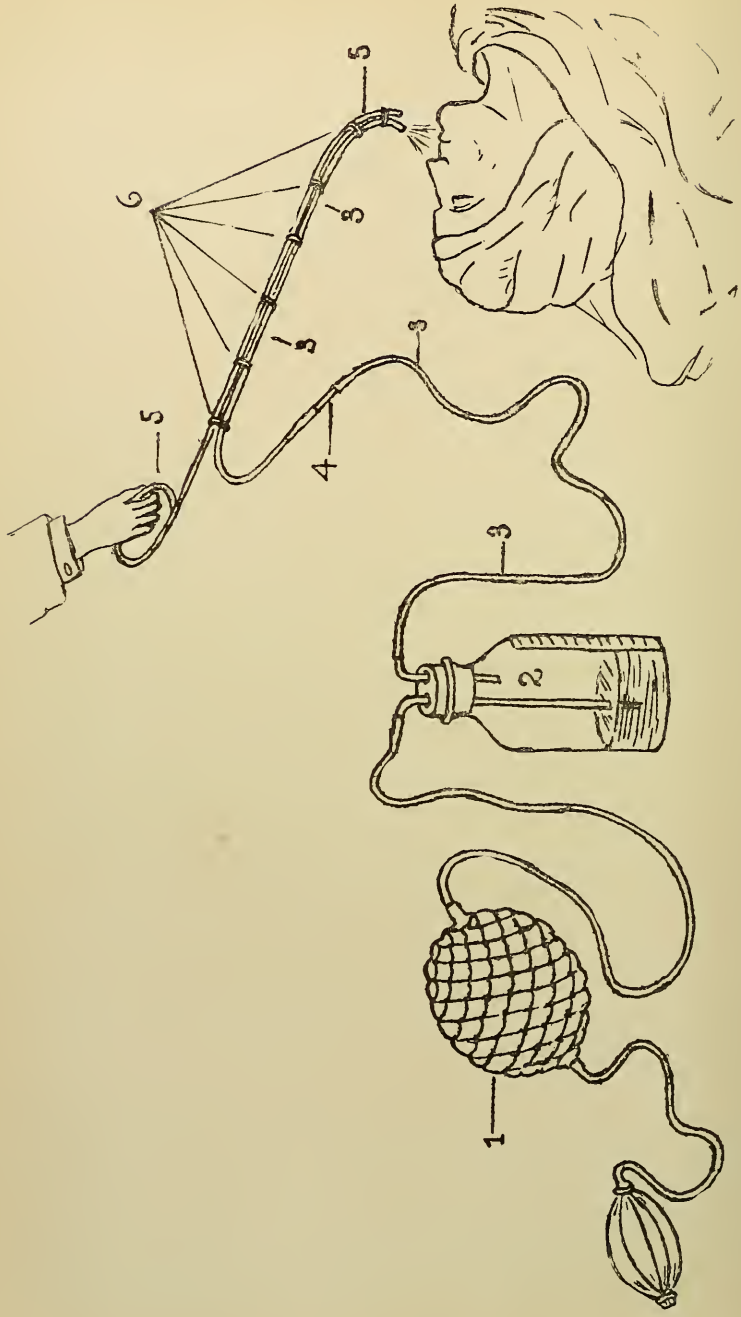
The now obsolete method of rectal etherization. The intra-nasal, or pharyngeal method, by which the ether or chloroform vapor is forced into the pharynx through a tube of rubber or glass passed into the nostril or mouth of the patient. The vapor is thus carried into the pharynx by a current of air which is forced through the ether or chloroform reservoir by the compression of a double bulb or bellows. In European hospitals the "Junker" apparatus, or some modification, is commonly used. In this country the anesthetic vapor is passed into the mouth through a glass tube, bent so as to fit the angle of the lips, or through a rubber catheter placed in the nose.

An improvement in these methods is that devised and used by Dr. T. Fillebrown,[1] the advantage of whose apparatus is that the ether vapor is inhaled without any part of the apparatus being brought in contact with the patient. This method possesses the desirable features enumerated above, but the apparatus, as described by Dr. Fillebrown, is of such a character that although quite convenient when once in the possession of the operator, still is not readily obtained without the assistance of a dealer in dental or surgical supplies. Also, it is somewhat bulky for transportation. For ordinary work a more simple appliance is desirable.

To obtain this the apparatus to be described was devised and used by the writer. It has thus far been used only with ether. It has been used for prolonged operative work in cases such as incision of the jaw and tongue with ligation of the carotid artery for carcinoma, excision of the parotid, cleft palate and hare-lip operations, trephining and intracranial exploration in head injuries, resection of the infra-orbital nerve, et alii. It has shown such utility that its publication seems justified.

The apparatus consists of a double bulb and rubber tube such as is used with a Paquelin cautery (1); a 12-oz. glass bottle fitted with a cork and glass tubes (2); a rubber tube

[1] A New Apparatus for Continuing Anesthesia while operating on or in the Mouth. By Dr. Thomas Fillebrown, M.D., D.M.D., Boston, U. S. A. Dental Cosmos, December, 1895. The essentials of the Fillebrown apparatus, to which the writer's attention was kindly recalled by Dr. George H. Monks, have been considered in the apparatus described in this article.


5
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2
1

of such length as will conveniently reach from the bottle to the patient's face (3); a short piece of glass tube (4) to connect (3); a piece of fairly heavy, moderately flexible copper wire (5); several rubber rings (6) (narrow sections cut from the end of tube (3) and used to fasten (3) to wire (5)). These are combined in the manner shown in figure.

To use this apparatus the bottle is partly (about one-third) filled with ether. At the proper time the patient's end of the "delivery" tube (3) (previously sterilized) is connected with the other portion of the apparatus by means of the glass tube (4). The wire (5) supporting the delivery tube (3) is so bent that the ether vapor escaping from it can be directed towards the patient's mouth and nostrils without impeding the surgeon in his work, the hand of the etherizer grasping it at the loop, while he stands behind or at the side of the patient. The ether vapor is then forced through the tube (3) from the bottle (2) to the patient's face by squeezing the bulb (1) with the other hand in the same manner as a Paquelin cautery point is kept incandescent.

It has been found by experience that it is usually a rather slow and difficult process to commence the anesthesia with this apparatus, although in a majority of cases one can keep a patient anesthetized in a satisfactory manner after he has once become unconscious. It is more rapid and satisfactory to etherize a patient in the usual way, and to substitute the modified apparatus when the patient is on the table ready for operation. When so used the sterilized portion of the apparatus is not brought into use till the operation field is clear. Now the only portion of the apparatus which comes near the patient's face is the sterile end of the delivery tube (3). This should not touch it. The etherizer at the other end of the wire (5) cannot unintentionally come in contact with the aseptic site of operation.

The efficiency of the apparatus is increased by having a good air space above the surface of the ether in the bottle. Also by placing the bottle in a basin of warm water, or wrapping it in a hot cloth.

The usual distance between the mouth of the patient and

the end of the ether tube has been about three inches. It has not been found necessary to place it in contact with the patient. If the mouth is held open with a gag the effect of the ether is more marked. It is also more convenient to control the amount of oral secretions by atropia.

A strong current of air in the room blows the ether away from the patient and may interfere seriously with its effect.

The etherizer must pay close attention to his task. In cases where the method works efficiently the ether is supplied in such quantities that a dangerous narcosis can be produced unless the operator realizes what the apparatus is doing. It is often possible to suspend the current for several minutes without the patient's regaining susceptibility to pain. With care, however, there is no danger.

The susceptibility of patients seems to vary with the individual, as is observed with the usual methods of etherization. It is more easily and satisfactorily used with children than adults, possibly because they are affected by smaller amounts than adults. "Alcoholics" show the same power of resistance as by other methods and in some cases have required large amounts.

In some cases a medium sized glass funnel (as is used for filtering solutions) inserted in the patient's end of the delivery tube (3) seems to increase the efficiency of the apparatus. It is held slightly nearer the face of the patient than the end of the tube when used without it.

The average amount of ether used by this apparatus is five to six ounces hourly. Four ounces and fifteen ounces are the maximum and minimum amounts used per hour. In favorable cases the person familiar with the apparatus will be able to maintain a satisfactory condition of anesthesia with a very small amount of ether after the patient is once thoroughly anesthetized.

No freezing effect, or irritative action on mucous surfaces, has been noticed, but care has been taken not to direct the vapor too long continuously upon the same cutaneous area.

The results of the writer's experience and observation are the following conclusions:

That the method is not successful in all cases.

That it requires close attention on the part of the etherizer; more so than is absolutely necessary, but not more than is desirable, with the usual etherization, which at times is wrongly regarded as a trivial affair.

In favorable cases it is very satisfactory.

It affords the surgeon an unobstructed field of operation.

He can work continuously and uninterruptedly.

The face of the patient is freely visible so that the patient's condition is readily noted. An important factor in case of a careless etherizer.

The etherizer is not in the way of the surgeon or assistants. He can do his work at arm's length. His distance from the patient is regulated by the length of the wire carrier (5) of the delivery tube.

The administration of the ether is continuous.

There is no danger of wound infection from the ether apparatus, since it does not come in contact with the patient.

In a large proportion of the cases of operation on the head and neck where this method has been tested it has been satisfactory.

The apparatus is easily obtained and simple in construction.

The writer, in conclusion, wishes to acknowledge the courtesy of his colleagues by which the opportunities for testing this form of apparatus have been greatly increased.

X.

THE NORMAL APPENDIX: ITS LENGTH, ITS MESENTERY AND ITS POSITION OR DIRECTION, AS OBSERVED IN SIX HUNDRED AND FIFTY-SIX AUTOPSIES.

By George H. Monks, M.D., and J. Bapst Blake, M.D.

For a number of years the Pathological Department at the hospital has been in the habit, while making any autopsy, of observing and recording certain facts about the vermiform appendix in all cases, even where no pathological conditions existed in connection with it. This was done at the direction of Drs. Councilman and Mallory, with the idea that sooner or later the facts thus recorded might be of use to the Surgical Department. It occurred to us, therefore, on looking over the records, that there was already a sufficient number of cases to furnish, when tabulated, definite information as to the length of the appendix and its mesentery, and the frequency with which the appendix lay in its different positions, about which points there has been some diversity of opinion among the various anatomical and surgical writers. Upon application to the Pathological Department the records were placed entirely at our disposal, and we decided to consider all cases, except those in which the cause of death could be traced directly or indirectly to some trouble in the appendix.

Of the 1,400 autopsies recorded between January 10, 1896, and May 29, 1902, we found that a note had been made as to the appendix in a large proportion of them. Omitting from this number the cases in which the appendix bore an etiological relation to the cause of death, there remained some 656 cases of men, women and children

indiscriminately, in which some mention or record was made of the appendix. We give under each heading a few extracts from well-known authorities before presenting our figures.

THE LENGTH OF THE APPENDIX.

Quain[1], quoting Berry and Rannshoff, gives the average length 92 mm. (three and one-half inches), and the extremes 31 mm. (a little more than one inch), and 230 mm. (a little less than nine inches).

Gray[2] agrees with Quain exactly.

Treves[3] gives the average length as four inches, and the extremes one and six inches.

Deaver[4] gives the average as about four inches, and the extremes as one-half inch and nine inches.

In the series of cases we tabulated the measurements were originally all made in centimetres, and for this reason they were probably more accurate than if made in inches. In tabulation we have used considerable care to take nothing for granted, except what is spoken of as being assumed.

In the 641 autopsies of our series, in which the length of the appendix is recorded, we found the following:

Average length .	7.90 cm.	(three inches).
Shortest . .	1. cm.	(a little over one-third of an inch).
Longest . .	24. cm.	(a little over nine and one-third inches).*

RELATION BETWEEN THE LENGTH OF THE APPENDIX AND SEX, AGE AND BODY LENGTH.

There does not appear in our figures any apparent difference in the length of the appendix in the two sexes. Children, however, by these same figures, have a proportionately greater length of appendix than adults. Treves says that the appendix obtains its full length quite early in life.

Other than this, the appendix does not bear any necessary relation to body length. The longest appendices in our series, and also the shortest, were found generally speaking in bodies of medium length. The converse is also true

* Nine and one-third inches is almost exactly the height of this page.

that the longest and shortest bodies do not necessarily have the longest and shortest appendices.

RELATION BETWEEN THE LENGTH OF THE APPENDIX AND RACE.

All the subjects of autopsy were white, except 23 negroes, 3 Chinamen, and 1 "brown Hindoo." Of the last no length of appendix was given. These cases are too few to allow of any generalization. The average appendix length in the 26 bodies was, however, 8.2 cm. (approximately our general average).

MESENTERY: ITS PROPORTIONATE LENGTH.

Quain says that the appendix has a mesentery for more than half its length. The distal portion is generally quite free.

Gray says that the distal third or so is free from mesentery.

Treves says the mesentery "may extend to the tip of the appendix in the fœtus, but in the adult it often only reaches to the centre of the tube, or to the junction of its middle with its distal third."

Deaver says that it occupies "the entire length (as I have usually found it)." It is, however, "at times absent."

Of the 277 cases of our series, in which mention is made as to the mesentery (meso-appendix), we find the following notes, most of them indicating what proportionate distance, measured from the base of the appendix, was supplied by mesentery.

	Per cent. of 243 cases, where measurement is given.
No mesentery	16 cases (6.5%)
Mesentery reaching less than ½ the length of the appendix:	
¼ 3	
⅓ 5	
"small" 1	
—	9 cases (3.7%)
Mesentery reaching ½ the length of the appendix	16 cases (6.5%)

Mesentery reaching more than $\frac{1}{2}$, but not for the entire length of the appendix:		
$\frac{2}{3}$	29	
$\frac{3}{4}$	15	
$\frac{4}{5}$	1	
	—	45 cases (18.5%)
Mesentery reaching (practically) the entire length of the appendix:		
"entire"	128	
"for entire length"	23	
"almost entire"	1	
"almost for entire length"	3	
"complete"	1	
"$\frac{10}{11}$"	1	
	—	157 cases (64.6%)
Mesentery present, but no particulars given		34 cases.
		277 cases.

POSITION AND DIRECTION OF THE APPENDIX.

Quain speaks of the principal directions of the appendix as being (in order of frequency):

1. Up and to left,
2. Down and to left,
3. Into pelvis,

and says that the appendix is sometimes entirely behind cœcum.

Gray quotes various authorities, all of whom differ.

Treves says: "In the majority of cases in the adult body the appendix, when examined *in situ*, will be seen to lie behind the end of the ileum and its mesentery, and to point in the direction of the spleen. In the only other position that may be said to be at all common, the little tube ascends vertically behind the cœcum."

Deaver says: "In the majority of cases the appendix will hold one of eight positions. Dr. Bristow suggests a very simple method of classifying these positions and directions, which consists in locating a central point in the right iliac fossa which, in its most common position, will represent the attachment of the appendix to the cœcum, and from this central point lines are drawn that radiate in eight different directions." He goes on to say that Fowler substituted the

points of the compass for the numbers used by Dr. Bristow. He also states that the central point is a point from two to two-and-one-half inches on a line from the anterior superior spine of the ilium in the direction of the umbilicus. Deaver, following Fowler's suggestion as to the points of the compass, states that the appendix is most commonly found in one of the following five positions:

1, N.E.; 2, S.E. or S.; 3, N., to right of colon; 4, N. or N.E., in front of colon; 5, under the cœcum, generally N.

We found some note referring to the position and direction of the appendix in 572 cases of our series. We assume that the position of the cœcum was in the right iliac fossa in all these cases, unless it was otherwise noted, and that the direction as given corresponded to a line from "a central point," similar to that referred to by Deaver, as above. In some of the cases position only is referred to, as "behind cœcum," "in pelvis," etc. These we have thought best to place with the others where direction only was spoken of, but under distinctive headings.

When our cases had been tabulated we found that most of them divided themselves naturally into eight groups, indicating eight different directions from a common centre, and it was not until after we had arranged our diagram that we learned we had been anticipated by others in this method of demonstration. As, however, the nomenclature used by us is somewhat different from that before used in connection with the diagram, we shall later reproduce the latter, as we believe it still to be the best method of graphically recording the different directions in which the appendix may point.

In the following tables we have preserved, so far as possible, the diction used in the original records. Such words, however, as "upwards," "downwards," "inwards," etc., have been shortened to "up," "down," "in," etc.

The groups have been arranged in such a way as to indicate the order of frequency of the different positions and directions.

Down and In: (The most common position or direction.)

"Down and in"	54
"Hanging over pelvic brim"	116
"Behind cœcum, down and in"	6
"Inner side of cœcum, down"	1
"Below cœcum, and in"	1
"Loops into pelvis, tip just under mesentery of ilium"	1
	179

Behind Cœcum: (The second commonest position; * direction not given.)

"Curled / Coiled up behind / beneath cœcum"	68
"Behind cœcum"	32
"Posterior to cœcum"	2
"Back"	1
"Curled behind cœcum at level of costal border"	1
	104

Down: (The third commonest position, or direction.)

"Below cœcum"	37
"Behind cœcum, down"	13
"Down"	8
"Caudad"	5
"Curled up below cœcum in right iliac fossa"	4
"Below cœcum, down"	2
"Directed down"	2
"Down and back"	2
"From anterior surface of cœcum passes directly back"	2
"Directed down, appendix in close contact with abdominal wall"	1
"Down, back and up"	1
"Down, back and up, folded on itself"	1
"Below and behind cœcum"	1
	79

*It is, of course, true that the great majority of appendices are, *at their origin*, behind the cœcum. If we were to add, therefore, to the class "behind cœcum" all those which, though originating there, eventually assumed a definite direction, that class would stand first of all. We have not done this, because the full descriptions recorded in each case seemed to us to justify the present classification.

In: (The fourth commonest position or direction.)

"Toward median line"	53
"Inner side of cœcum, curled up"	2
"To median line"	1
"Toward median line, 4 cm. from end of cœcum, par. with ileum for 5 cm., then turns back"	1
"Behind cœcum, in"	1
"In and slightly back"	1
"Extends to median line at pelvic brim"	1
"Inner side of cœcum, inwards"	1
"Behind loop of small intestines"	1
	62

Up.

"Up behind cœcum"	40
"Up and back"	5
"Up"	3
"On top of cœcum"	2
"In front of cœcum, directed up"	1
"Posterior to cœcum, cephalad"	1
	52

Up and In.

"Up and in"	23
"In and up behind cœcum"	6
"Inner side of cœcum, up and in"	3
"Up, back and in"	3
"Up and in for half its course, and doubled on itself"	1
"Up and in, passes over anterior surface right kidney to within 1 cm. of upper margin, closely united to renal capsule"	1
"Points almost towards umbilicus"	1
"Behind ileum and cœcum"	1
	39

Up and Out.

"Up, behind and to right of cœcum"	7
"Up and outside (or outer side of) cœcum"	6
"Up and out"	6
"Up and out, behind cœcum"	3
"Up and out, close to iliac crest"	1
"Up and out, over anterior surface of cœcum"	1
"Up, back and out"	1

"Up and slightly back on right of ascending colon"	1
"Up, outside attachment of cœcum"	1
"Outer side of cœcum, extends up"	1
"Outside cœcum, up and doubled twice upon itself"	1
	29

Out.

"Out"	2
"Behind cœcum, out"	2
"Behind and to right of cœcum"	2
"Outside of cœcum, along parietal wall"	1
"Curled up on itself, exterior to cœcum"	1
"Back and out"	1
	9

Down and Out.

"Behind cœcum, down and out"	2
"Below and to right of cœcum"	2
"Down and out"	1
	5

In Pelvis (unclassified).

"Wholly within pelvis"	4
"Free in pelvis"	3
"With cœcum in pelvis"	3
"Within pelvis"	1
"In pelvis, directed upwards"	1
"S-shaped loop in pelvis"	1
"Deep in pelvis"	1
	14

While we do not consider this classification ideal, we deem it the best one under the circumstances. Several of the cases might possibly be classed under some other heading, but these cases are mostly small in number, so that, if they were so placed, they would not materially influence the result.

Reverting to our diagram we find that in more than half (320) of the cases the appendix points within the right lower quadrant (that is "in," "down and in" and "down"). If now we add to this number those which are classed simply under

the heading "behind cœcum" without any direction being given (104), we find that in 424 of our total of 572 cases, or in 74 per cent., the appendix is either "behind the cœcum" (without specified direction), or it points "in," "down," or "down and in," the last being by far the most frequent position or direction of all.

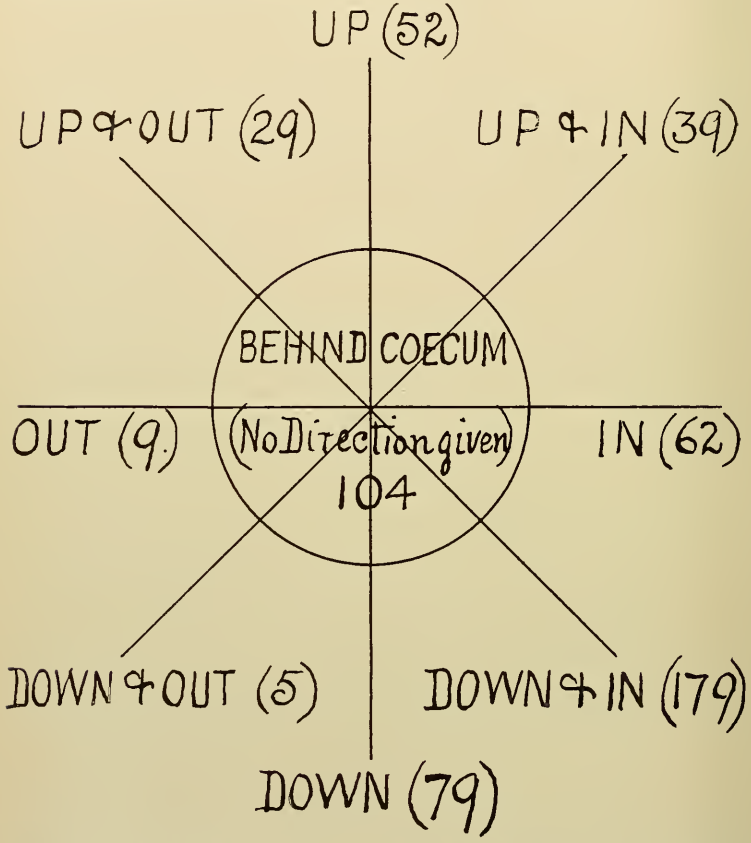

Diagram Showing the Different Positions and Directions of the Appendix in 558 Cases, and the Actual Number of Cases Occupying each Position or Direction.

UNUSUAL CASES.

(*A.*) *Conical Cœcum.* — There were nine cases where it was recorded that the cœcum tapered into the appendix.

(*B.*) *Fibrous Cord.* — There were six cases where the appendix was represented by a white, glistening, fibrous cord (obliterated appendix). In an additional one the distal portion only was obliterated.

(*C.*) *Miscellaneous.* — One appendix "is directed up behind cœcum; passes over anterior surface of right kidney to within about 1 cm. of its upper margin."

Another is "curled up behind cœcum which is situated high up just below costal margin."

A third extends "up and in, the tip lying close to gall-bladder."

A fourth extends "up externally to the right border of the liver."

A fifth lies "between the right lobe of liver and right kidney."

A sixth lies "behind peritoneum under cœcum, and extends upward to lower border of right kidney."

A seventh lies "transversely across abdomen, slightly upwards, reaching beyond the middle line."

Conclusions.

From the foregoing statistics the following conclusions would appear to be justified:

(1.) *Length.* — The average length of the appendix in men, women and children, indiscriminately, is *7.9 cm.* (or *3 inches*); the extremes being *1 cm.* (*a little over 1-3 of an inch*), and *24 cm.* (*a little over 9 1-3 inches*).

There is apparently no relation between the length of the body, the sex or age of the subject, and the length of the appendix, except that children are apt to have an appendix proportionately longer than adults.

(2.) *Mesentery.* — Fully one-half of all appendices have a mesentery (meso-appendix), which reaches nearly to, or quite to, the tip. Of the other half by far the greatest proportion have mesenteries reaching as far as the middle point of the appendix, or beyond it. Occasionally an appendix has no mesentery at all.

(3.) *Position (or direction).* — The commonest position (or direction) for the appendix is "*down and in*," (*i.e.*, toward the pelvis), the appendix very frequently hanging over the brim. The second commonest position is "*behind the cœcum.*" * The third is "*down*," and the fourth is

* See foot-note on page 129.

"*in.*" *The appendix is in one of these four positions in about three-fourths of all cases.*

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1. Quain. Elements of Anatomy, 10th edition. 1896.
2. Gray. Anatomy, American edition from 13th English edition. 1896.
3. Treves. Lectures on the Anatomy of the Intestinal Canal and Peritoneum in Man. Brit. Med. Journal, 1885, p. 415.
4. Deaver. A Treatise on Appendicitis. 1896.

XI.

ERYTHRAGGLUTININS IN A CYST FLUID.

By Walter Remsen Brinckerhoff, M.D., First Assistant in Pathology, and Elmer Ernest Southard, M.D., Second Assistant in Pathology.

[From Pathological Laboratory, Boston City Hospital.]

Bordet, '99, pointed out that theories of agglutination must take account, not alone of bacterial agglutination, but also of the agglutination of blood corpuscles and of casein-particles.

Malkoff, '00, worked upon the agglutination of red blood corpuscles, and showed that the clumping substance has specific combining affinity for the element clumped, and, furthermore, in a normal serum which clumps different cells there are as many different specific agglutinins as there are species of cells clumped. Malkoff employed the corpuscles of the goat, against which he immunized guinea-pig and pigeon.

Flexner and Noguchi, '02, studied the effect of venoms upon agglutination, as well as upon lysis, and pointed out that the red corpuscles of the rabbit are highly susceptible, whereas the corpuscles of the guinea-pig, dog, sheep, swine and ox, are less susceptible in that order.

The most recent paper we have seen is that of Ford, '02. Ford chose an agglutinin occurring in normal serum (that in rabbit serum for hen's corpuscles), produced its anti-agglutinin, produced by immunization a second agglutinin of rabbit's serum for hen's corpuscles, and then a corresponding anti-agglutinin. He found that two anti-agglutinins were interchangeable and therefore identical. He concludes that the body obtained by immunization is merely quantitatively different from the body found normally.

The following notes concern a fluid from a non-malignant parovarian cyst removed at operation. The fluid was found to dissolve and to agglutinate the red blood corpuscles of the rabbit. The fluid was heated for one half hour at 54°–59° C, whereupon hæmolytic power was lost.

Clumping was obtained of the washed corpuscles of the normal rabbit, of rabbit immunized to the cyst fluid, of dog, and of one man. Other human corpuscles, including those of the patient, were never agglutinated. Agglutination of the guinea-pig's corpuscles was so slow as to be valueless for experimentation.

Upon repeated intraperitoneal injections of the cyst fluid, amounting in toto to 50 c.c., a rabbit was found to yield a serum having the property of delaying or inhibiting the agglutination of its own and other rabbit corpuscles.

The serum of the patient is markedly haemolytic for rabbit corpuscles. Upon inactivation by heat no agglutinative power could be demonstrated.

The fluid (Cy) was prepared by heat for agglutination work (Cy—C, where C is the complement). Saline suspension of washed corpuscles of the rabbit (wcR) was placed in the same amount in a series of test tubes. To each was added a different amount of Cy—C, *e.g.*,

wcR+(Cy—C) gtti,
wcR+(Cy—C) gttii, etc.

That series of tubes was found in which notable clumping occurred where the observed drop was taken from the hollow of the tube, *e.g.*,

NaCl+wcR+8, (briefly for [Cy—C] gttviii),
naCl+wcR+16,
naCl+wcR+32.

With this series was taken the limiting negative,

naCl+wcR+4.

The whole series was centrifugalized. The supernatant fluids, thus,

naCl+4,
naCl+8—x,
naCl+16—x,
naCl+32—x,

were applied in the same amount to fresh wcR. These supernatant fluids proved to cause agglutination in similar ascending series. Thus, where

8—x=y, then
16—x=y+8, and
32—x=y+16.

The quantities suggested can be only qualitatively confirmed, owing to the nature of the phenomenon of agglutination. Thus

wcR+naCl+8—x

gives no reaction, or a border-line reaction, and

wcR+naCl+16—x<
wcR+naCl+32—x.

SUMMARY.

The foregoing may be summed up as follows:

The cyst fluid exhibits

1. Hetero-erythragglutinins for
   (*a.*) rabbit.
   (*b.*) dog.
   (*c.*) guinea-pig (delayed).
2. Iso-erythragglutinin for one man.
3. No other iso-erythragglutinins in cases examined (no auto-erythragglutinin).

The cyst fluid is capable of developing an anti-erythragglutinin.

The patient's serum, though equally hæmolytic with the fluid, shows no agglutinative power.

The rabbit erythragglutinin sustains a quantitative relation to the amounts of corpuscles agglutinated.

A definition of the type of agglutinate obtained in work like the above may serve to bring out more clearly the value of the blood corpuscles in the study of agglutinins. The kind of aggregate observed may be shortly termed poikil. agglutinate in contrast with the homoagglutinate or rouleau.

Slight acquaintance with the red corpuscles shows that they clump in two forms, in one of which the arrangement

might be simply termed streptic, in the other staphylic. But these terms are scarcely definite enough, as further examination shows.

In the streptic type they adhere rim upon rim to form the coinrows or rouleaux. In the staphylic type they adhere by prominent angles upon surfaces of apposition which are accidental. In the rouleaux the elements preserve to a degree their biconcavity. The corpuscles may remain biconcave in the staphylic aggregates, but they may be variously crenate, vesicular, or otherwise distorted. The elements of a rouleau are alike, whereas the unlikeness in the elements of a tridimensional clump may be striking. Thus the streptic agglutinate and the clump in three dimensions might be more strictly termed, to employ the usual prefixes, homoagglutinate and poikilagglutinate.

Nevertheless, though the elements of a rouleau tend to show biconcavity, the elements of the poikilagglutinate (such as one gets in experiments like the above) may be equally normal-looking. Though the streptic agglutinate may be said to depend upon shape of elements with change of surface or change of ambient fluid, the staphylic agglutinate is formed without regard to element shape, consistently with great variety in surface character of its units, and in the absence of its usual medium.

Striking molar changes in surface, certainly involving marked molecular redistribution, are consistent with the constant and regular occurrence of poikilagglutination. But these changes are so many and so various (crenation, vesicularity, distortion, laking tendency) that they can hardly be invoked as the cause or necessary condition of the clumping.

The phenomena of corpuscular clumping thus serve to demolish in a simple manner those theories of agglutination which regard surface changes as the essence of clumping in general. That demolition had indeed been long since practically accomplished; but the blood corpuscles seem an especially fitting weapon.

The foregoing study emphasizes once more the quantitative character of those phenomena of which haemagglutination is one representative, sets forth a certain advantage in

studying the blood corpuscles to make clear the nature of agglutination, and indicates the desirability of further work upon agglutinins and similar principles of natural occurrence in pathological fluids — a sort of casuistics in immunity work.

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XII.

PYLEPHLEBITIS AND LIVER ABSCESSES FOLLOWING APPENDICITIS.

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Portal infections following appendicular inflammation are not infrequent, and although the condition is spoken of by all writers as rare, still it is a complication that may confront the practitioner in any case of appendicitis. It is with a view of emphasizing the comparative frequency with which this condition may be met that the appended cases have been studied. In this article especial attention has been given to the pathological processes, *i.e.* the gross findings at autopsy, the condition of the appendix in operated cases, the histology of the involved viscera with special reference to the liver lesions, and the organisms found in connection with the process.

The clinical picture of this condition has frequently been presented. Even before the recognition of appendicitis as a distinct malady, cases nearly identical in type with the ones here described were reported.

Loison attributes to Waller (1846) the first description of a case in which perityphlitic inflammation was followed by pus in the portal vein and liver abscesses; and Berthelin cites a case described by Hillairet in 1849 of illeo-cæcal abscess surrounding the appendix and all the hepatic veins in the liver filled with pus. In 1854 Buhl cited a similar case in which there were multiple liver abscesses, the largest the size of a hen's egg.

Since that time the condition has become well known and many cases have been collected. Loison calls attention to the groups of cases collected (independently) by Fitz, Langheldt, Einhorn, and Caley. These include in all twenty-nine cases out of six hundred and sixty-nine appendicitis autopsies. Bärensprung in 7,326 autopsies (Berlin 1859–73) found one hundred and eight cases of liver abscesses, eight following affections of the cæcum or appendix. Dudley found in 28,034 patients at the Zurich Clinic, twelve cases of liver abscesses, two following appendicitis. Kobler in 17,204 autopsies (Vienna 1881–90) found seventy-nine cases of liver abscesses, three following appendicitis; and in 1,307 autopsies at Sarajevo were ten liver abscesses, none following appendicitis. Borchardt found five cases in three hundred and ninety-eight autopsies. Davidson reports sixty-one cases of liver abscesses, nine with pylephlebitis in which the cæcum or appendix is given as the point of departure of the infectious process. Harry Jackson has reported sixteen cases of liver abscesses, in ten of which he gives appendicitis as the source of the subsequent infection.

Hart reports twenty-eight cases of liver abscesses occurring at the Presbyterian Hospital (N. Y.) in seventeen of which the infection was through the portal vein; three of these had their origin in an inflammatory process in the appendix. Musser gives an analysis of thirty-four cases of liver abscess of children under fifteen years of age. The abscesses were secondary to a pylephlebitis in four cases; to a pyæmia twice. Appendicitis as a primary cause is mentioned in one of the latter cases. Munro has reported nine cases of portal infection following appendicitis, most of which were confirmed at operation or autopsy. Four of these cases are included in the present article.

Reports of single cases of this condition are numerous and all present points of interest. Loison cites a number of these cases, among which are those of Fränkel, Bezançon and Netter showing multiple small abscesses of the liver and those of Peyne, Schoemaker, DeGennes, MacClelland and Rabé, showing one large, together with many small abscesses. Loison's own case, which resulted in recovery, was a case

with single liver abscess, as was probably a case with recovery reported by Sheen and a similar case of Munro's reported by Burrell and Bottomly.

Trowbridge reports a case in which a shawl pin was found in the appendix; termination liver abscesses and death. Ashby reports a similar case of a pin in the appendix. The appendix showed acute inflammation, and near it was a ragged ulcer. The liver contained three large abscesses. Church reports a third case with pin in the appendix, pus in the portal vein and two liver abscesses. In the case of MacClelland previously referred to a pin was also found in the appendix.

Krakowitzer reports a case in a boy eleven years old in which an abscess behind the colon was followed by sloughing of the appendix. There was peritonitis on the right side, emboli in the vessels of the liver and numerous liver abscesses. Lockwood mentions two cases, and Southey, Stooke, Jorand, Colquhoan, Dale and others have reported cases nearly identical with the type cases of this article, namely, ulceration of the appendix, pus in the portal vein and its radicles, multiple abscesses of the liver.

Four cases autopsied at the Boston City Hospital last summer together with four additional cases occurring at the same institution within the last few years, in which appendicular infection was followed by a suppurative inflammation of the portal vein, liver abscesses, septicæmia and death, have furnished the material for the present article.

These cases presented at autopsy the following pictures:

G. M. (97.286). Male, 30 years old. Well-developed man but emaciated. Shows slight jaundice.

Abdominal cavity. About 200 cc. of brownish cloudy fluid present. Old adhesions of great omentum to right iliac wall. Appendix and coecum bound down by old adhesions also. Mesenteric glands enlarged and soft, mesentery generally thickened.

Spleen wt., 475 gms. Large, rather soft; capsule smooth. Malpighian corpuscles not visible; pulp much increased.

Liver wt., 2,565 gms. Large. On anterior surface could be seen many small whitish patches, especially on left lobe. On under surface of left lobe were areas of multiple abscesses, one about 6 by 5 cm. in diameter, near the upper left border, and another near inferior border of same lobe. On under surface of right lobe many whitish spots. Section

showed abscess formation, especially in left lobe, portal vein was dilated and filled in part with rather firm yellowish masses, and in part with pus. On opening the portal vein and its branches they are found occluded by yellowish adherent thrombi and thick yellow pus.

Appendix bound down by old adhesions, canal patent, filled with a mass of pus; mucous membrane roughened and ragged, showing evidence of old inflammation. Other organs not remarkable.

Anatomical Diagnoses.

Chronic appendicitis.
Suppurative pylephlebitis.
Multiple abscesses of liver.
Chronic peri-appendicitis.
Abscesses of mesenteric glands.
Acute splenic tumor.

C. H. (U.00.16) Autopsy, May 24, 1900.

Age 52 years. Small emaciated man. Peritoneal cavity contains no fluid. Serosa of intestines smooth, glistening; slightly injected. Appendix 5 cm. long, directed inward; proximal half bound down posteriorly by old fibrous adhesions. Lumen of distal half is completely obliterated. Mesenteric lymph nodes not palpable.

Liver wt., 1,250 gms. The common bile duct is normal. The gall bladder is normal. The right border of the liver and the adjacent two-thirds of the anterior and posterior surface of the lobe are dark reddish brown in color. The remainder of the surface is reddish yellow. The demarkation between the two is very sharp. The lesser omentum is severed and the portal vein found to be nearly occluded by a large red thrombus firmly adherent to the vessel wall. The diameter of the portal vein at this point is about 1 cm. Traced downward towards the mesenteric roots of the portal vein, the thrombus gradually becoming smaller and passes into the superior mesenteric vein for a short distance. The lining of the lower portion of the portal vein and of the mesenteric vein appears normal. Within the liver substance the portal vein becomes dilated to a diameter of about 1.5 cm., and filled with a dark grayish puriform fluid. The liver on section is of normal consistence. The lobules can be made out with difficulty. The branches of the portal vein are filled with a rather thick pus. The walls of most of the branches can be made out, and apparently are not involved in the suppurative process. The surrounding liver tissue, the smaller bile ducts, and hepatic artery appear unchanged. Only a few branches of the portal vein are found that are not involved. These are chiefly in the left lobe. The upper portion of thrombus, already described in the portal vein near its entrance to the liver, has undergone puriform softening.

Other organs not remarkable.

Anatomical Diagnoses.

Suppurative pylephlebitis.
Thrombus of portal and mesenteric veins.
Chronic obliterating appendicitis.

Eld. (U00.17). Autopsy, May 26, 1900.

Male, 21 years of age, Conjunctivae slightly yellow. Abdominal incision only allowed.

In peritoneal cavity there is about 250 cc. of yellow seropurulent fluid, rather thin, and having a faecal odor. This is all through the cavity, but principally in the upper part. Peritoneum is dull and in places covered with shreds of fibrinous exudation. There are about the spleen a few slight fibrous adhesions. Coecum is firmly bound to the lateral wall by fibrous adhesions. After separating these for a distance of about 3 cm. the stump of the appendix was found. It is about 5 cm. in length, and is closely surrounded by fresh fibrous adhesions. Just below this a small walled-off pocket, with thick yellow pus, is found. There is not more than $\frac{1}{2}$ cc. of this pus; smears from which show numerous pus cells and a few streptococci. Over the surface of the right half of the liver are numerous yellow areas, some of which are soft, and in the left lobe is an abscess cavity of considerable size. The veins about the mesentery, the coecum and the lower end of the ileum show no signs of thrombosis. Vessels of the lower abdominal parietes in this region are injected, but there is no sign of any thrombus in the veins. Portal vein is free from thrombus except within 2 cm. of the liver.

Spleen wt., 180 gms.

Liver wt., 1,980 gms. It extends below the costal border about 6 cm. Surface is covered with some shreds of fibrin. Dark bluish red ; vessels injected. Over the whole surface are yellow areas, most of which are nearly round ; they vary from .5 cm. to 3 to 4 cm. in diameter. About these yellow areas there is a dark bluish red irregular border. In the upper posterior part of the right half there is one of these yellow areas which is raised over .5 cm. above the surface, and beneath it there is considerable fluctuation covering a space of 5 by 7 cm. Over the rest of the right half of the liver these yellow areas are slightly raised, on an average of 2 to 3 cm. in diameter, and many of them are quite soft. Occupying a good part of the left lobe of the liver is a cavity, irregular in shape, about 15 by 12 cm. In the thin upper wall of this cavity is a round opening a little over 1 cm. in diameter. It has a yellowish necrotic border, and through this the purulent contents of the abscess has escaped into the peritoneum. On the under surface of the liver these abscesses are not so numerous ; only one is ruptured. On transverse section through the liver there is not more than one-half of the normal liver tissue. The rest of the area is occupied by irregular abscess cavities which are a little more numerous in the upper two-thirds. The liver tissue is dark reddish brown ; centers of lobules dark red. Tissue is quite moist, and near the abscess walls it has a bluish green color. These abscesses are lined with a thin glistening fibrous wall. They are irregular, with numerous small sacules, and many of them communicate with one another. They contain a thick light yellow granular pus. Numerous small hard orange colored particles of inspissated bile. The portal vein as it enters the liver is nearly occluded by a thrombus. This grayish red necrotic mass extends up on one wall of the portal vein, and after it has entered the liver about 1 cm. there is a large gray necrotic mass lining the opposite wall. On opening up the branches

all the veins running to the liver contain small thrombi all the way through the liver tissue, and a great many of the veins found to communicate with the abscess cavities.

Gall bladder moderately filled with greenish bile containing small thrombi all the way through the liver tissue, and a great many of the veins found to communicate with the abscess cavities.

Gall bladder moderately filled with greenish bile containing orange yellow particles. Gall duct is open ; normal. Bladder wall is not apparently thickened.

Intestines opened only in one place ; contents normal.

Anatomical Diagnoses.

Multiple abscesses of liver.
Acute purulent peritonitis.
Abscess near appendix.
Thrombosis of portal vein.
Pylo-phlebitis of portal vein.
Acute splenitis.

M. S. (00.32). Autopsy, February 27, 1900.

Male, 42 years old. Abdomen contains 1,700 cc. of slightly turbid amber fluid. Slight fibrinous deposit on peritoneum in pelvis. Coecum adherent to abdominal wall along the line of the scar. Appendix, coecum and beginning of ileum bound together by fibrous adhesions. Posterior to coecum and appendix is a small abscess containing a thick yellowish pus. Retroperitoneal lymph nodes much enlarged, and several of them are completely softened. Along the spine to the left of median line is a vessel .75 cm. in diameter, probably left spermatic, with thickened walls, containing a pale red thrombus easily removed.

On section anterior to the foramen of Winslow the portal vein is seen to be filled with yellowish pus and its walls considerably thickened. Passing in around and slitting open the vein this condition is seen to extend downward a considerable distance along the attachment of the mesentery. Inferior vena cava normal. In the transverse meso-colon, along the pyloric portion of the greater curvature of the stomach, are several small collections of thick yellowish pus, the largest 2 cm. in diameter. These are probably in veins of the portal system. Lesser peritoneal cavity appears normal. Vessels of mesentery on section half-way between base of mesentery and small intestine appear normal, blood flowing from the cut ends.

Spleen wt., 275 gms. Purplish red and of about normal consistency. Here and there are ill-defined paler areas, the largest 1 cm. in diameter.

Gastro-intestinal tract. Stomach filled with a greenish opaque fluid, apparently containing considerable pus. Near pyloric end are two small ulcers, the largest 1 cm. in diameter. The edges are elevated and slightly reddened. On slight compression a considerable amount of pus is forced out. Upper part of small intestine contains considerable dark green fluid material. Mucosa normal. Appendix 6 cm. long. The end is bulb-like, consisting apparently of dense fibrous tissue. Mucosa of colon is pale and unbroken. In rectum are two small ulcers similar to those in stomach,

Liver wt., 1,910 gms. Brownish red. Here and there on the cut surface are groups of slightly elevated yellow areas, the largest 0.75 cm. in diameter. On section the same color is presented. Lobular markings distinct. Interlobular areas very pale. Distributed irregularly throughout the liver are numerous abscess cavities, the largest about 2 cm. in diameter, containing a thick, slightly greenish pus. Smears, stained by Gram and methylene blue, show numerous pus cells, but no bacteria. Gall bladder bound to the surrounding structures by rather firm adhesions. It is small, and contains thick bile.

Kidneys wt., 375 gms. On gentle pressure pus exudes from numerous points on the cut surface of left kidney in the columns of Bertini, and between the cortex and pyramids, and to a less extent in the cortex. Smears show numerous pus cells, but no bacteria. Pelvis slightly injected. One of the vessels, apparently the renal vein, is filled with pus and its wall is thickened.

Genital system. Prostate on section presents a few ill-defined reddish areas. On gentle pressure pus exudes from a number of points immediately surrounding the transverse section of the prostate, apparently cross section of veins. Pus may be pressed from similar points in various parts of the pelvic organs.

Anatomical Diagnoses.

Acute sero-fibrinous peritonitis.
Acute peri-appendicular abscess.
Thrombosis of left spermatic vein.
Acute suppurative pylephlebitis.
Acute abscesses of lungs and kidney.
Ulcers of stomach.
Ulcers of rectum.
Multiple acute purulent abscesses of liver.
Acute purulent phlebitis of left renal vein.
Acute purulent phlebitis of hæmorrhoidal and vesico prostatic plexuses.

H. C. B. (01.110.) Autopsy, June 5, 1901. Male, age 35 years. Skin generally has a slight icteric tint, and conjunctivæ are distinctly yellow.

Peritoneal cavity. Greater peritoneal cavity; peritoneal surface is lustreless. All the coils of bowel are slightly adherent by easily broken fibrinous shreds. In the cavity of the pelvis is a small amount of sero-sanguenous fluid. At a point two metres from the cœcum several short coils of the ileum are firmly adherent, requiring some force to separate them. No volvulus made out. The portion of the gut involved in this mass is 60 cm. in length, and its surface is everywhere covered with fibrinous shreds. In the region of the appendix the appendix stump is found, and on pressure nothing escapes from the lumen of the cœcum. Between the spleen, stomach, left side of diaphragm, liver, and posterior wall of peritoneal cavity is an abscess containing offensive yellowish semi-fluid, purulent material. Its wall is everywhere covered by a fibrinous membrane, about 1 cm. in thickness, which is readily detached. Mesenteric lymph nodes all somewhat

enlarged. One mesenteric lymph node is softened, and contiguous to it is a canal (mesenteric vein), with roughened yellowish walls admitting the little finger, which communicates directly with the portal vein. The contents of this canal is a sputa-like, yellowish muco-purulent material. The portal vein, both outside and inside of the liver, is greatly dilated and filled with the same material above described. The primary branch of the portal vein is 3 cm. in diameter. The splenic vein contains a red, rather friable clot. The hepatic, cystic and common ducts are normal and pervious. Likewise the gall bladder.

Spleen wt., 295 gms. The organ is flabby and deeply indented along each of its borders. On section, color of a gray red.

Gastro-intestinal tract: Normal throughout.

Liver wt., 1,740 gms. On the inferior surface of the left lobe are two small rough holes, 3 to 4 mm. in diameter and 2 to 3 mm. deep; on pressure yellowish muco-purulent material exudes from this opening. On section, throughout the left lobe, the portal veins are dilated, and contain yellowish sputa-like material. In the region of the superficial losses of substance above described, the liver tissue, for a short distance about them, is firm and yellowish. In the right lobe, particularly towards its superior surface and towards the right, are numerous yellowish, softened, irregular areas, arranged in clusters, .3–.5 cm. in greatest diameter. On pressure, material like that found in the portal vein exudes from surface of these areas. The intervening liver substance is brownish red in color and of normal consistence. The hepatic vein, so far as can be opened, is normal. Primary branches of portal vein are much dilated, but their walls are smooth.

Smears from contents of portal vein and from subphrenic abscess show streptococci flattened and staining by Gram. Some in cells.

Other organs not remarkable.

Anatomical Diagnoses.

Subphrenic abscess. Left.
Mesenteric and portal pyophlebitis.
Acute general peritonitis.
Acute splenic tumor.
Multiple abscesses of liver.
Chronic appendicitis.

T. A. T. (01,145.) Autopsy, Aug. 15, 1901. Male, age 17 years.

Autopsy performed through abdominal incision.

Peritoneal cavity contains a small amount of clear fluid. Most of peritoneum is smooth, glistening and free from adhesions. The mesenteric lymph nodes are enlarged, varying from .5 to 2 cm. in diameter, and are grayish pink on section. About the appendectomy wound, between abdominal wall and lower end of cœcum, are numerous adhesions. Hidden away in these is the stump of appendix (appendectomy about three weeks previously). Along the ascending colon, and particularly between hepatic flexure and neighboring parts of abdomen, jejunum, liver, gall-bladder and stomach are numerous adhesions. These are easily broken apart and consist of young connective tissue and fibrin.

The walls of the above-mentioned hollow viscera are also considerably thickened in this region. On tearing the cœcum from its attachments several small pockets of grayish brown, thick pus are opened into. Behind the cœcum is a large lymph node (3 cm. in greatest diameter), which has a caseous (in part calcified) centre. Another node higher up has a soft purulent centre. Branches of the superior mesenteric vein running from the lower part of the ascending colon are occluded by rather friable clot mottled grayish-red and gray in color. These are only slightly adherent, but above, in the main superior mesenteric vein, especially that part behind the pancreas, the clot is pale in color and firmly adherent. In the portal vein is grayish-brown, slightly viscid purulent fluid. Other branches of portal system seem to contain red clots.

Spleen wt., 280 gms. Is large, smooth, dark red, except area at one end, about 5 cm. in various diameters, which is lighter red, with a few areas of yellowish gray. On section dark red. Malpighian bodies not very distinct.

Gastro intestinal tract. Stomach normal. Small intestine shows many large areas of congestion and submucous haemorrhages. Colon hyperaemic. In region of hepatic flexure is a deep punched out ulcer about 2 cm. in greatest diameter. This has rather overhanging edges and its base is formed by softened necrotic looking tissue suggesting a broken down lymph node. In connective tissue about this node (if it is one) are several stitches. (At recent operation purulent lymph nodes in this region were incised and drained.) About 8 cm. from this towards coecum is a second deep punched out smaller but similar ulcer.

Liver wt., 1,570 gms. Surface smooth. On section numerous small areas of bright yellow color against a brownish gray background, at places becoming reddish. From many periportal spaces, especially in right lobe can be squeezed whitish creamy pus. Some of these abscesses are 1 cm. in diameter, but the great majority are 2–3 mm. Smears from this pus show leucocytes and a small number of slender Gram staining bacilli.

The branches of the portal vein at hilum of liver contain dirty grayish pus. Gall bladder and ducts normal. Hepatic vein normal.

Anatomical Diagnoses.

Localized adhesive peritonitis.
Pus pockets and softened lymph nodes about coecum.
Thrombosis of superior mesenteric vein and branches.
Suppurative pylephlebitis with multiple abscesses of liver.
Acute splenic tumor.
Ulcers of colon.

D. S. (01.185.) November 7, 1901. Male. Age 16 years.

Skin everywhere shows a marked icteric tinge.

Abdominal cavity. The intestines are lusterless and very slightly injected in places, but there are no definite signs of peritonitis. Their adjacent surfaces are very slightly adherent, being extremely easily separated as if held together by a slight viscidity of serosa. Passing through the operation wound the fingers enter a small walled-off cavity, the walls being formed by the coecum, the small intestine and the parietal peritoneum. In this pocket is the stump of the appendix. The

lumen is patent, and from it can be squeezed brownish faecal material. The loops of intestine forming these walls separate with difficulty. The pelvic cavity is normal. Between the liver and the diaphragm are numerous firm, fibrous adhesions. The adhesions are also marked between the gall bladder and colon, between colon, gall bladder, and duodenum, and between the liver and right kidney. The gall bladder is distended with clear serous bile stained fluid. Common bile duct, hepatic and cystic ducts opened in situ and are everywhere patent. Pancreatic duct patent. The liver extends 12 cm. below the tip of ensiform cartilage in the median line. Over the anterior surface of the right lobe are numerous light yellow irregular areas .5 to 1.5 cm. in diameter. Abdominal contents removed *en masse*. Kidneys are dissected out and retro-peritoneal tissue dissected away until portal vein is reached. Portal vein opened towards liver and in opposite direction to mesenteric veins. Veins of ilio-coecal valve, to middle point of small intestines, to spleen and to stomach opened to a point of juncture with each viscus. All contain a rather thick dark grayish red fluid. In the upper part of the vein (towards the liver) this fluid becomes lighter in color and more distinctly purulent. The vein going to the appendix region differs in no way from the other branches. The walls of the veins are everywhere smooth and show no change. The incision towards the liver is carried into the veins going to both the right and left lobes of the organ. Both veins are filled with blood-stained pus. The hepatic vein and inferior vena cava are normal.

The ilio-coecal and mesenteric lymph nodes are enlarged, largest measures 1.2 by .5 by .5 cm. On section they are grayish pink. Consistency normal. The nodes in the region of the foramen of Winslow are larger, measuring 2 by 2 by .75 cm. They show nothing remarkable on section.

Liver wt., 3,265 gms. (after escape of considerable pus from abscesses) Right lobe firmly adherent to diaphragm and hepatic flexure of colon. Left lobe of normal color; not adherent. Right lobe dark green with numerous irregular light yellow patches .5 to 1.5 cm. in diameter. On cutting through these light areas a creamy yellow pus exudes. On section the right lobe is found to be honey-combed with abscesses of varying size, the largest being 3 cm. in diameter. These abscesses are most numerous in the central and anterior portions of the right lobe and here almost coalesce to form a single large cavity. The extreme right is free from them, but the normal liver markings can not be made out. In the left lobe the process is confined to the veins which are filled with yellowish purulent material. The liver tissue encircling the veins is soft and greenish colored. Smears from the abscess cavities show a small Gram decolorizing bacillus, a large Gram staining bacillus and cocci, singly, in pairs, and in short chains staining by Gram. Other organs not remarkable.

Anatomical Diagnoses.

Pylephlebitis (suppurative).
Multiple abscesses of liver.
Chronic fibrous perihepatitis.
Acute splenitis.
Appendictomy.

E. B. (01.188). Autopsy, November 10, 1901. Female, age 11 years. Fairly well developed, much emaciated girl. Conjunctivae icteric. Beginning just below the costal margin, 5 cm. to the right and parallel to the median line, is an open wound 10 cm. in length, containing a ganze drain which extends down behind the liver; also a scar 2 cm. in length, just above and parallel to Poupart's ligament.

Peritoneal cavity. Serosa appears normal. Omentum is long, devoid of fat. Its lower end is bound down to the coecum and small intestine in the appendix region by easily separable fibrinous adhesions. Separating these adhesions a small pocket is found containing the necrotic end of the appendix adherent to the coecum. Just behind this is a small irregular opening into the coecum, through which faecal matter can be expressed. The tissues about are necrotic. In the pelvis is an area 5 cm. in diameter, including the posterior portion of the uterus, the rectum, broad ligament and adjacent peritoneum, which is covered with a thick layer of yellowish tenaceous fibrin.

Mesenteric lymph nodes appear normal, with the exception of one in the ileo-coecal region, which is 2 cm. in longest diameters, has a yellowish, irregular surface, and is caseous.

On opening the portal vein it is found to be occluded by a soft, purulent, adherent thrombus, which extends up to the bifurcation into branches going to the right and left lobes of liver; from this point to smaller branches in liver the vein is filled with bile-stained purulent material — walls of vessels smooth. This same process extends throughout the upper portion of the superior mesenteric vein, and through its ileo-coecal branch to the appendix. Its other branches are normal. The splenic vein is normal and not occluded, as are also its branches, the gastric, pancreatic and the inferior mesenteric veins. The biliary and pancreatic ducts are normal.

Liver wt., 1,000 gms. (after drainage of considerable pus). The surface is smooth, covered with innumerable small gray dots against a dark red background. Over the centre of the anterior surface of the right lobe this marking is of a greenish hue. On the anterior surface of the lower part of the left lobe is an aperture 2 cm. in length, 1 cm. in breadth, the edges of which are ragged, and covered with bits of yellow fibrin. (Operation wound for drainage.) On section both right and left lobes are honey-combed with abscess cavities from .5 to 2.5 cm. in diameter, containing thick, yellow pus, which exudes when they are cut. The greater confluence of these cavities is in an area a little to the left centre of the right lobe, involving substance throughout a space as large as the hand, which appears almost as one large abscess. A similar area, but a little smaller, is seen in the lower posterior portion of the left lobe.

Between these abscess cavities the liver substance shows a red background, with numerous minute patent blood vessels, around which there is a sharply circumscribed zone, grayish-yellow in color, making areas $\frac{1}{2}$ to $\frac{3}{4}$ mm. in diameter. There are also similar areas in which no blood vessels can be made out.

Anatomical Diagnoses.

Pylephlebitis (suppurative).
Multiple abscesses of liver.
Adhesive peri-appendicular peritonitis.
Pelvic abscess.

SUMMARY OF PROTOCALS.

It will be seen from the above that all cases except one showed numerous small (multiple) abscesses in the liver, more frequently greater in number in the right lobe, and averaging in size from 2 mm. to 5 cm. in diameter.

Three cases showed, in addition to the small abscesses, large cavities preferably selective for the upper anterior part of the right lobe.

One case was remarkable in the fact that there were no abscesses in the liver, though the branches of the portal vein in the liver substance were filled with pus, and the increased connective tissue in the organ seemed to show the process to be of some duration.

In all cases the portal vein was filled with pus or occluded by purulent thrombi. The mesenteric veins were involved in five cases, in one case the vein running to the appendix being everywhere filled with pus, while the other veins of the mesentary were normal. (Loison points out that in most cases the veins show involvement, but in some cases the organism may travel the vein, setting up the liver suppuration, without leaving demonstrable changes in the vessel.)

One of the cases showed two small ulcers in the colon, and one case showed two small ulcers in the stomach and colon. These cannot be excluded as a possible source of infection, though that the process originated in the appendix in both these cases is the more probable conclusion.

The spleen was enlarged in five cases. Jaundice present in five cases.

Gall ducts were patent and normal in all cases and pancreatic ducts normal in all cases where they were examined.

No.	Liver.	Veins, etc.	Appendix.	Complications.	Spleen.	Bact.
97.286.	Multiple abscesses largest 5x6 cm., throughout left lobe. Few small abscesses in right lobe.	Portal vein and branches filled with pus and adherent thrombi.	Chronic and acute processes both present.	Abscesses of mesenteric glands.	Enlarged and soft.	B. Coli Communis.
U00.16.	No abscesses, but portal vein and its branches throughout the liver are dilated and filled with pus.	Portal and superior mesenteric veins thrombosed.	Lumen obliterated. Old adhesions.		Normal size.	B. Coli Communis. Staph.Pyog.Aureus.
U00.17.	Multiple abscesses ¼ to 4 cm. throughout right lobe. Large cavity, 15x12 cm., left lobe.	Thrombosis of portal vein beginning 2 cm. from liver.	Appendix abscess. (Operation.)	Acute purulent peritonitis.	Soft, but not enlarged.	Streptococcus Pyog. B. Coli Communis.
00.32.	Multiple abscesses averaging 2 cm. scattered throughout liver. (Both lobes.) No large single cavity.	Portal vein and veins of meso-colon filled with pus.	Chronic inflammation and peri-appendicular abscess.	Small ulcers in stomach and rectum. Abscesses in lungs, kidneys and prostate. Peritonitis.	Enlarged.	Streptococcus Pyog. B. Coli Communis.

No.	Liver.	Veins, etc.	Appendix.	Complications.	Spleen.	Bact.
01.110.	Multiple abscesses very small, .3–.5 cm., especially in upper part of right lobe and inferior surface of left lobe.	Portal and splenic veins contain pus.	Chronic appendicitis. (Operation.)	Subphrenic abscess. Acute general peritonitis.	Enlarged.	Streptococcus Pyog. B. Coli Communis.
01.145.	Multiple abscesses .3 to 1 cm. throughout both lobes. No large cavity.	Veins of portal system contain pus and thrombi.	Acute appendicitis. (Operation.)	Ulcers of colon. Pus pockets about cæcum.	Enlarged.	B. Coli Communis.
01.185.	Multiple abscesses $\frac{1}{2}$ to 3 cm. throughout both lobes. Thickest at centre of right lobe.	Veins of portal system filled with pus.	Acute appendicitis. (Operation.) Cavity walled off.		Enlarged.	Streptococcus Pyog. B. Coli Communis.
01.188.	Multiple abscesses $\frac{1}{2}$ to $2\frac{1}{2}$ cm. throughout both lobes. Focus of cavities at centre of right lobe.	Portal vein and its branch to appendix filled with pus.	Acute appendicitis. (Operation.) Peri-appendicular peritonitis.		Normal size.	Streptococcus Pyog. B. Coli Communis.

APPENDIX.

Appendictomy was done in five of the cases, either late in the course of the disease or after recurring attacks. Three of the cases did not come to operation. Of the three latter cases, one showed a chronic appendix inflammation, and the other two showed both chronic and acute processes. Of the operated cases, two of the appendices were examined at the time of operation.

(01.110.) Stump of appendix, 2.5 cm. long, serosa covered with rather tough fibrinous adhesions, wall slightly thickened, 3 mm. Lumen a little dilated, mucous showing some injection. *Microscopic.* Showed portion of mucosa fairly well preserved, muscularis slightly thickened and infiltrated with numerous lymphoid and plasma cells. No evidence of acute inflammation.

(01.185.) Appendix, 5.5 by 1.5 by 1.3 cm., buttress mesentery. Sorosa diffusely red, muscularis slightly thickened, mucosa red and sodden. *Microscopic.* Appendix infiltrated with numerous polymorphonuclear leucocytes. Foci of hemorrhage. Acute appendicitis.

This suggests that no relationship exists between the pathological changes in the appendix and the severity of the subsequent infection. A portal pyo-phlebitis is liable to follow a slight destruction of the mucosa, a complete perforation, or, in fact, any inflammatory condition of this organ.

HISTOLOGY OF THE LIVER.

Sections of the liver, hardened in Zenker's fluid, imbedded in paraffine, and stained by Eosin-Methyline blue, Mallory's connective tissue stain and by Gram-Weigert stain, were examined and showed as follows:

(97.286.) The sections show changes everywhere. The vessels are dilated, and in the portal spaces there are large accumulations of plasma cells. Around some of the spaces there is marked formation of connective tissue. In the capillaries there are large numbers of cells; definite polynuclear leucocytes, plasma cells and the large transitional

forms. There are several abscesses in the section. In the neighborhood of the abscesses the liver cells are atrophic. The more definite abscesses show a mass of fibrin and bacteria in the centre, around which is an area of polynuclear leucocytes, and much nuclear detritus. None of the abscesses have a definite wall, but they extend directly into the liver tissue.

(00.16.) None of the sections show abscess formation within the liver epithelium, but there are well marked central necroses, fairly evenly distributed. The liver cells of these areas are mostly homogeneous, and in places leucocytes and endothelial cells are seen invading the liver cells. The portal veins are dilated, and filled with leucocytes, fibrin and a few red blood corpuscles. The periportal connective tissue is increased and infiltrated with plasma cells. The bile ducts are dilated.

(00.17.) No sections.

(00.32.) In liver there are a great number of small and large abscesses, and in some branches of the portal vein there are purulent thrombi. The abscesses represent definitely circumscribed masses of pus cells in fairly good preservation. The adjacent liver cells are compressed and elongated, and some of them necrotic. In the liver, at some distance from the abscesses, there is marked hyperplasia of the capillary cells, with atrophy of the epithelium. No emboli of organisms seen.

(01.110.) Increase of connective tissue about all portal spaces. Liver cells not stained. An area separated from liver by connective tissue containing bile ducts, in which are many cells. The cells are mostly polynuclear leucocytes, many of which are degenerated, and phagocytic cells. Areas show marked vacuolation of the liver cells.

(01.145.) There is a very extensive necrosis which involves almost the entire lobule. Not infrequently liver nuclei are in process of indirect division. This indicates some attempt at repair. Most of the liver cells show large fat vacuoles. In the periportal connective tissue are numerous polynuclear leucocytes. These can be traced only a short distance into the lobule. Some of the portal veins are

filled with leucocytes and fibrin threads. In the lumen of some of the bile ducts are polynuclear leucocytes.

(01.185.) Section shows many small abscess cavities, the boundaries of which are not sharply circumscribed, but which show the leucocytes infiltrating more or less deeply and irregularly into the surrounding liver epithelium. In a few of these areas the centres are broken down, and stain sharply with eosin. There are many areas of congestion, the red blood corpuscles extending out between the liver cells, and not being confined exclusively to the capillaries. The periportal connective tissue shows everywhere focal collections of leucocytes, and in places the leucocytes can be seen infiltrating the space between the liver cells. The liver cells in these areas are small, irregular, and granular, and stain with eosin. Foci of organisms can be seen with the oil immersion in the necrotic material, in the centres of the abscess cavities, and can also be found both within and without the cells most remote from these areas. In no place in the section is a perfectly normal picture presented.

(01.188.) The liver is congested. The liver cells are swollen, reticulated, and stain with eosin. Small abscesses are seen filled with leucocytes and broken-down cells. The section contains very little normal liver epithelium; everywhere are areas of cell infiltration — or degeneration and vacuolization of the liver cells starting apparently from the centres of the liver lobules. The nuclei of the cells are fragmented, and at times numbers of these fragments are seen included in large pale cells. Small collections of organisms can be seen in some of the cells.

Briefly summarizing, we see that in nearly all these cases the greater part of the liver tissue shows marked degenerative changes. The parenchyma, except in one instance, is riddled with numerous small abscesses which show no evidence of self limitation. These abscesses are surrounded by liver cells which present various degrees of degenerative change. Areas of necrosis are also seen apart from the abscesses. Masses of organisms can be seen both at the site of the abscesses and among the best preserved liver cells. The portal radicles are distended with pus or occluded by

thrombi. The bile ducts are dilated, and the periportal connective tissue is increased in amount, and infiltrated with plasma cells and leucocytes. In only one instance is an attempt at repair evident.

BACTERIOLOGY.

In the reports of cases previously referred to little or nothing is said concerning the organisms found in connection with the process. Loison found in pus taken from his liver abscess a number of organisms, including staphylococci, streptococci and colon-bacilli, and Lockwood describes the appendix in his case as infiltrated with cocci, diplococci and streptococci. From the present cases cultures and smears were obtained at autopsy, and in two instances sterile swabs inoculated at the time appendectomy was performed:

Examination of these latter showed the following:

	Culture.
01.110	B. Coli Communis.
01.185	Streptococcus Pyog. B. Coli Communis.

In one case (01.145) culture was taken from the liver at time of operation. This showed Staph. Pyog. Albus and B. Coli Communis.

At autopsy cultures and smears were taken from the liver and portal vein in six cases. Cultures alone in one case, and in one case smears alone.

LIVER AND PORTAL VEIN.

	Culture.
97.286	B. Coli Communis.
01.145	———
u.00.16	B. Coli Communis. Staphylococcus Pyog. Aureus.
u.00.17	B. Coli Communis.
00.110	B. Coli Communis.
00.32	Streptococcus Pyog.
01.185	Streptococcus Pyog.
01.188	Streptococcus Pyog.
01.145	Gram decolorizing bacillus in smear.

In sections of the liver stained by Gram-Weigert's stain the organisms can be seen in the necrotic material of the abscess cavities, and can also in some cases be found within and without liver cells most remote from these areas.

As would naturally be expected, the organisms found are those associated with the usual cases of appendicitis, namely, for the most part the streptococcus and the colon bacillus. (Streptococci are found in about 90 per cent. of the routine appendix cases examined at this laboratory.) It is to be noted that they are here present in five of the eight cases. Out of the three remaining cases there are two in which only incomplete examinations were made. In the third case the Staphylococcus Pyog. Aureus is found.

It is interesting to note in this latter case (00.16) that though the veins in the liver are everywhere filled with pus the sections fail to show abscess formation within the liver parenchyma, while in the streptococcus cases the liver is riddled throughout with numerous small abscesses. This latter is probably merely a coincidence, as guinea pigs inoculated into a mesenteric vein with either staphylococcus, albus or aureus show liver abscesses in three or four days.

It may be said, therefore, that the organism found in the appendix throws no light on the subsequent course of the disease. The appendix merely acts as a culture tube for the development of the infectious organism. The importance of the early removal of this source of infectious supply is obvious, especially as the veins of the appendix have comparatively large lumens, and their anatomical relation to the rest of the portal system is calculated to conduce veinous engorgement. Moreover, the absence of valves allows clots to spread.

CLINICAL HISTORY.

A detailed clinical history has been given in most of the reported cases, and the history of four of the present cases has already been published by Munro, so that it is unnecessary to speak of it here at length. Suffice it to say that while a fairly typical case of appendicitis, followed by chills, irregular and severe, a continued high temperature, pain and

tenderness over the trunk of the portal vein and the liver, jaundice and enlarged spleen in a proportion of the cases, and marked emaciation may lead to an easy diagnosis, still any one or group of these symptoms may be absent, thus rendering diagnosis extremely difficult. Dr. Munro admirably summarizes the symptoms as follows: "More or less, perhaps fleeting jaundice, irregular chills, hepatic tenderness and emaciation should suggest a portal pyophlebitis following appendicitis, present or remote.

PROGNOSIS.

The hopelessness of this condition may be seen by a glance at the autopsy findings in these cases. While in an occasional case we get a mild infection or a single abscess, the evacuation of which, surgically or spontaneously, offers a chance for recovery, still the general tendency is towards multiple abscess formation. Thus the liver is converted into a huge purulent sponge which renders surgery impotent.

In the rare cases of recovery the disease is a tedious one, covering a period of months. The patient, not only being subjected to several severe surgical operations, but also reduced to a skeleton by the pyæmic process, rarely is able to resume his vocation before the end of six months.

TREATMENT.

Early recognition of the condition, followed by prompt surgical measures — the removal of the inflamed appendix and free exploration and thorough drainage of the liver — offers the only chance of recovery.

CONCLUSIONS.

Abscesses of the liver following appendicitis and portal infection are usually multiple and selective for the anterior-superior surface of the right lobe, though they may be found anywhere throughout the organ.

The pathological process in the appendix and the infectious organism differ in no way from the process or organism found in uncomplicated appendix cases.

This condition is far more common than is generally sup-

posed and the potentiality for this pyæmic sequence should be kept in mind in connection with every patient who has, or has had, appendicular inflammation.

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XIII.

EPENDYMAL EPITHELIUM AS A CONSTITUENT OF A TERATOMA.

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PRIOR to 1895 our knowledge of neuroglia tissue was very incomplete. Almost simultaneously in this year appeared the publications of Weigert[1] and Mallory.[2] In these, very similar staining methods are given for the demonstration of neuroglia fibres, and both observers note that the minute dots or rods, previously described by Weigert[3] as occurring in the border of the epithelial cells of the ependyma, have the same microchemical reactions as the neuroglia fibres. Subsequent to these, numerous studies of neuroglia, both in normal and pathological conditions of the central nervous system, and in gliomata, have appeared, and other staining methods of distinct value have been added, notably Mallory's phosphotungstic acid hæmatoxylin stain.[4] Apparently these methods, however, have not been applied to tumors occurring elsewhere than in direct connection with the central nervous system, with the exception of the case of subcutaneous glioma over the coccyx reported by Mallory before the American Association of Pathologists and Bacteriologists, March 28 of this year; nor have the dots described by Weigert in the ependymal epithelium been noted in tumors except by Mallory[5] in gliomata.

Weigert. Beiträge zur Kenntniss der normalen menschlichen Neuroglia, Frankfurt a. M., 1895.

[2] Mallory. Centralbl f. allg. Path. u. path. Anat., 1895, vi., 753.

[3] Weigert. Anat. Anzeiger, 1890.

[4] Mallory. Jour. of Exp. Med., 1900, v., 19.

[5] Mallory. Jour. of Med. Research, 1902, vii., 1.

The presence in teratomata of elements of the central nervous system, in a more or less completely developed state, has frequently been reported, and often especial attention has been directed to their occurrence as throwing some light on the still much disputed question of the origin of such complex tumors.

In this connection descriptions are given of neuroglia and ependymal epithelium. These are often incomplete, and frequently the author indicates his doubt as to their true nature in some such way as glia (?), neuroglia (?), glia-like network, ependymal-like epithelium, or structures suggesting choroid plexus. Even since the publication of microchemical reactions for neuroglia these conditions have continued, and in a review of the literature of teratomata from 1895 to date no case was found in which these methods have been applied to the study of neuroglia and ependymal constituents of such tumors.

To a teratoma of the ovary, received through the kindness of Drs. Brown of Newburyport and Munro of Boston, the modified fibrin and the phosphotungstic acid hæmatoxylin stains of Mallory were, among others, applied.*

The teratoma had developed in the ovary, as shown by the presence in sections of ovarian stroma containing ova in direct continuity with tumor tissue. Of the total bulk of the tumor about half is made up of a large cyst; the remainder is solid. Its structure is complex, and all three germ layers are represented. The solid part contains innumerable cysts ranging from very minute ones to those measuring 1 cm. in diameter. Of the tissue between the cysts almost half consists of a meshwork of fibres giving specific microchemical reactions of neuroglia. The remainder is made up of fibrous connective tissue containing a small amount of elastic tissue, smooth muscle, bone, cartilage and small islands of epidermis. In a few areas of neuroglia tissue nerve cells with Nissl's granules are found.

The structure of the cysts is varied. A few are lined by epidermis showing the usual keratin transformation with

*The clinical history of the case and gross description of the tumor are appended at the end.

here and there a hair follicle and sebaceous gland. Other cysts show a stratified transitional epithelium and many have a lining of ciliated cylindrical epithelium in which globlet cells are frequent. Less frequently than the latter are cysts lined by similar epithelium, but devoid of cilia. Some cysts show a low columnar or cuboidal epithelium with cytoplasm thickly studded with rod-shaped granules of brownish pigment very similar to those found in the retina. Similar pigment also occurs in some groups of connective tissue cells.

Besides the above types of cysts, two others occur. One is lined by rather low cuboidal epithelium and has a sharp line of demarcation from the surrounding stroma; the other is lined by high cylindrical epithelium whose basal border is often irregular and between which are sometimes found neuroglia fibres. These two types of cyst have this in common: the nuclei of their epithelium are situated toward the outer end of the cell, and in the cytoplasm of many near the inner border are seen very minute dots or rods which give the same microchemical reactions as do the neuroglia fibres. Sometimes there is but a single dot, at other times two or three, and often six or eight or even more, always near the inner border. These are seen perhaps most distinctly where the section is cut so as to give a view of the top of the cell. Here the dots are seen to occupy the central portion of the end of the cell and not infrequently to have a concentric grouping. None of these cells are ciliated, and the picture is very different from that produced by cilia. These dots are occasionally found in the cytoplasm of a cell embedded in neuroglia fibres.

The relation of these cysts to the neuroglia tissue varies. As already mentioned, the high cylindrical type of cells frequently stands in very intimate relation to neuroglia fibres. The cysts lined by the low cuboidal type may be entirely or in part surrounded by neuroglia, or none may be in its immediate vicinity. The latter cannot be considered as against the ependymal character of this epithelium, since the same conditions hold in the choroid plexus, where ependymal epithelium is present and yet no neuroglia is adjacent.

Weigert, who first described these dots staining in the same

way as neuroglia fibres, considered that they occurred only in ependymal cells. Other observers since then have concurred in this opinion. Their presence then in some of the cysts of this teratoma would give evidence of the ependymal character of part of the epithelium, a proof we believe never before adduced in this class of tumors. Granting this, and with the large number of areas consisting of a meshwork of neuroglia fibres giving their specific staining reactions, a large proportion of the tumor undoubtedly is referable to the anlage of the central nervous system.

In conclusion I wish to express my thanks to Dr. F. B. Mallory for very many useful suggestions.

Clinical History. B. W., female, age 17, single. The patient has had the ordinary children's diseases, and at ten scarlet fever. She has, however, never been very strong, and has complained often of being tired. Bowels have been very constipated. For more than a year she has noticed an enlargement of her abdomen, more marked on the left side. This has steadily increased since first noticed, and has been accompanied by pain, worse on the left side. Two years ago there was a slight menstrual flow, but none before this time or since.

Physical examination on admission to Anna Jaques Hospital, Newburyport, Mass., shows patient to be well developed and nourished. Abdomen considerably distended, slightly more markedly just to the left of the umbilicus. Outlines of an irregular mass, extending about three finger breadths above the umbilicus, can be made out. Mass is firm, smooth and tense. It is movable and flat on percussion.

Vaginal examination showed in anterior cul de sac a large hard mass, apparently part of and growing from the cervix uteri. This moves with the larger tumor. Tubes and ovaries could not be made out. A uterine sound could be passed only 1¼ inches, where the canal seemed to bend to the left.

Operation by Dr. Munro, assisted by Dr. A. B. Brown. Median incision. A large cyst of the left ovary, with a long pedicle, is found. The cyst lies on the right side and the uterus is pushed toward the left. There are many large vari-

cose veins in the broad ligament, and the tube is stretched to double its normal length. Flat broad pedicle of tumor, including broad ligament, tube and round ligament tied off with interlocking chromic gut ligatures. Raw edges sutured over with catgut. Abdominal wound closed in layers with catgut, chromic gut, and horsehair.

Patient stood operation well, and made an uneventful recovery.

Gross description of tumor. B. C. H., S02.162. — Specimen is roughly oval in form, $20 \times 12 \times 15$ cm. It is covered by a grayish white connective tissue capsule, forming a smooth surface. Firmly attached to one portion of the tumor a Fallopian tube, with its fimbriated extremity, can be made out. The tube is patent.

The tumor consists of two parts, a solid irregular smooth mass and a cyst wall. The cyst has been incised, but when distended readily admits both fists. In the cyst cavity there is a small amount of yellowish gelatinous fluid and communicating with it are other smaller cysts. At one point in the cyst wall there is a small mass of curled up hair, surrounded by small bits of grayish white, grumous material, which resembles the contents of a sebaceous cyst.

Section through the solid portion of the tumor shows irregular masses of tissue of varying size, form and consistence, separated by connective tissue trabeculæ. Everywhere there are small, smooth-walled cysts from 3 to 7 mm. in diameter. Widely distributed through the tissue are dots and streaks of dark blue black or intensely black pigment which, against a grayish background, give a marbled appearance to the mass. In many places there are spicules of bone. In others cartilage and adipose tissue appear. The entire cut surface has a somewhat gelatinous appearance, and from it can be scraped a sticky gelatinous material. No large pieces of bone occur, and nowhere is there any symmetry of arrangement.

XIV.

SECONDARY CARCINOMA OF THE LIVER, WITH REPORT OF A CASE IN WHICH THE LIVER WEIGHED 15,110 GMS. (33⅓ LBS.)

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AMONG 1,650 consecutive post mortem examinations recorded in the Pathological Laboratory of the Boston City Hospital, 60, or 3.63 per cent., showed carcinoma affecting some organ of the body. Of these 1,650 cases, 362 were under 20 years of age, so that the per cent. of carcinomatous involvement for adult cases was 4.65 per cent. Again this total of 1,650 cases includes 268 from the South Department, where only the acute infectious diseases, as scarlet fever and diphtheria, are treated. Deducting these there remain 1,382 cases, of which 4.34 per cent. were carcinomatous.

The liver was involved in 25, or 41.66 per cent., of the 60 cases of carcinoma. This represents 1.51 per cent. of the total number of autopsies, or 1.94 per cent. of those over 20 years of age. Of these 25 cases the carcinoma was primary in the liver in 5, and secondary either by direct extension or by metastasis in 20.

The percentage of liver involvement in this series of cases is apparently a small one. In 4,200 autopsies at Guy's Hospital, reported by White,[1] 126, or 3 per cent., showed carcinoma of the liver, and this corresponds with the figure given by Leichtenstern,[2] who in

[1] White. Tumors of the Liver. Albutt's System of Medicine, 1898, vol. iv.

[2] Leichtenstern. Clinical Aspects of Cancer of the Liver. Von Ziemssen's Cyclopedia of the Practice of Medicine, 1880, vol. ix.

6,019 autopsies found carcinoma of the liver in 174, or 2.89 per cent.

In 311 cases of carcinoma at the Zurich medical clinic reported by Siegrist,[1] the liver showed involvement clinically in 45 cases, and this by post mortem examination was corrected to 77 cases, or 24.7 per cent. of the total.

An analysis of the 20 cases of secondary carcinoma of the liver shows 11 males and 9 females. The ages ranged from 28 to 70, as follows:

28 years	1 case.
30 to 40 "	2 cases.
40 to 50 "	3 "
50 to 60 "	7 "
60 to 70 "	7 "

The duration of the disease, owing to the very indefinite character of the early symptoms, is difficult to estimate. Where determinable it was in most cases from 3 to 8 months, and in some much less. In only one case was one year with certainty exceeded, and possibly in another where there was a history of gall stone attacks during a period of 8 years.

Jaundice was present in one-half of the cases, and ascites in one-third. In only 4 cases did the spleen exceed the average normal weight of 171 gms. as given by Orth, and in these cases the enlargement was explainable as the result of a concomitant acute infectious process, to which in one case was added a malarial splenitis.

In the 20 cases the primary growth was in the stomach in 5, pancreas 4, large intestine and gall bladder, each 3, rectum 2, œsophagus, breast and prostate, each 1.

The picture presented by the liver in the different cases varied both as to the extent and character of the involvement. In 13 cases the liver surface was nodular, the nodules varying in size from 3 mm. to 6 cm., or even larger. Umbilication of the nodules often was present. The cut surfaces of the livers were thickly studded with carcinoma nodules, generally whitish in color, but frequently mottled

[1] Siegrist. Klin. Untersuchungen über Leberkrebs. Deutsch. med. Woch., 1888, xlv., 145.

by bile staining or foci of hemorrhage. The liver tissue between the nodules appeared normal or showed evidence of compression. In none was there more than a slight increase of connective tissue in the liver substance. In all of these cases all parts of the liver seemed to share equally in the diseased condition.

In 4 cases the liver showed only a few nodules. In one of these there was a single nodule in the right lobe, in another one in the left lobe; a third showed two nodules, and a fourth a few.

In 2 cases the liver was involved by extension in the immediate vicinity of a primary growth in the gall bladder, besides showing a few scattered metastases.

One case differed from all the others in having an almost perfectly smooth surface, and a very extensive diffuse growth in the liver tissue, with only here and there the formation of small discrete nodules. The involvement was more marked toward the periphery of the liver than at the centre, and in places assumed an indefinite wedge-shaped distribution. This liver weighed 3,400 gms.

The structure of the liver growths was that of the primary, in most cases adenomatous in type. The size of the liver showed great variation. When the involvement was slight the liver showed little change, but even in cases with extensive and apparently equal involvement there was great variation in size. About half showed considerable enlargement. In no case was there marked atrophy.

The weights were:

1,000 to 1,500 gms.	5 cases.
1,500 to 2,000 "	5 "
2,000 to 2,500 "	2 "
3,000 to 4,000 "	3 "
5,000 and over	4 "
Weight not given	1 case.

In one of the cases the liver weighed 8,100 gms., while another reached the enormous weight of 15,110 gms. The amount of abdominal space encroached upon by the liver in the former case is well shown in the accompanying pho-

tograph (Plate I.). The latter case, besides its enormous size presents other points of interest, and is here given in detail.

PLATE I.

Clinical History. — G. W., age sixty-two years, a bookbinder by trade, was born in Grisons, Switzerland. His family history presents no points of interest. His father, mother and a sister lived to an old age, dying without any signs of a malignant growth.

P. H. The patient came to America in 1868. In 1873 he had small-pox. With this exception there has been no previous illness. He has always been temperate in his habits, and denies venereal disease. Since he was about thirty years of age he has drunk occasionally a small amount of beer and smoked in moderation.

P. I. The present illness dates definitely from September, 1898. Prior to that period the patient seems to have had no symptoms, at least none suggesting the need of medical attention. At that time his physician was consulted for loss of strength, dyspnœa on exertion and swelling of the abdomen. Just how rapidly these symptoms had developed is not known. His physician, Dr. Clement, states that, when seen in September, 1898, his liver extended far below the umbilicus on the right side, and well down to its level on the left side. The surface of this mass felt smooth, except near the mid line, where there was an area about 5 cm. in diameter, which was distinctly elevated.

The patient was ordered to take a vacation, and while away from Boston was very sick with vomiting, headache and dizziness. At this time he was in bed for almost six months, and then was able to be up, occasionally working for a day. In May, 1900, he became confined to the house, his feet began to swell, and his abdomen continued to enlarge.

PLATE II.

He steadily lost weight, and became jaundiced. The size of the abdomen and the degree of jaundice has from time to time varied.

Until about August, 1900, constipation had existed, though relieved readily. On several occasions, about this time, small amounts of blood were passed by the rectum. This was at the time attributed to obstruction of the portal circulation.

On November 24, 1900, he was admitted to the service of Dr. Sears, at the Boston City Hospital, for observation. A physical examination on this date shows a poorly developed, emaciated, slightly icteric man. His radial arteries are atheromatous, his pulse regular. The upper limit of the heart is at the second interspace; the right border at the level of third rib, 5.6 cm. to the right of the median line; the left border at the level of the fifth rib, 13.2 cm. to the left of the median line. The apex of the heart is not made out. Its action is regular and no murmurs are present.

Lungs are of good resonance, except on the right side, below the mid scapular region, where there is an area of dulness. A few moist rales are present chiefly in the bases.

The liver flatness begins at the fourth rib, and extends downward to a point 2.5 cm. below umbilicus. A large part of the abdomen is occupied by an easily palpable tumor mass, whose lower edge can be felt 5 cm. below umbilicus. This edge is irregular and blunt. Beginning at the mid line it can be traced to the right in an outward and slightly downward direction for 5 cm., but from here it dips more perpendicularly toward the pelvis. From the mid point toward the left it can be traced outward and slightly upward to its intersection with the left mammillary line, where it again dips downward and outward toward the pelvis. (Plate II.) The surface of the tumor thus bounded is hard, with several smooth mounds and ridges that are easily seen. Some of these have an elastic feel as though of fluid under tension.

The abdomen is tense and protuberant. Below the tumor edge there is tympany, except in the extreme flanks. The girth at a point 10 cm. above the umbilicus is 105 cm.

Blood Counts.

November 8, 1900:	
Red blood globules	3,704,000
Leucocytes	8,600
Hæmoglobin	75%
Differential count of 200 leucocytes:	
Polymorphonuclear neutrophiles	45.5%
Small mononuclears	31.5%
Large mononuclears and transitionals	9.5%
Eosinophiles	13.5%
November 24:	
Red blood globules	3,952,000
Leucocytes	9,800
Hæmoglobin	50%
Differential count of 333 leucocytes:	
Polymorphonuclear neutrophiles	50.8%
Large and small mononuclears	38.7%
Eosinophiles	10.5%

Three days after admission patient returned to his home.

During all this time the patient's appetite remained good. He had a light general diet, with much milk. Occasionally there was distress after eating. Pain was never a prominent symptom, and often was entirely absent. Accumulation of fluid gave no trouble until the last few months of life, and then was most in evidence in the legs, which were frequently tapped, with much relief. Often as much as three quarts of fluid escaped during twelve hours. The abdomen during this time contained fluid, probably as much as it could accommodate. During this period his symptoms were such as could be explained by the mechanical pressure of an enormously enlarged liver on neighboring organs, combined with an increasing exhaustion. He died August 19, 1901.

Clinical Summary.

Previous history negative.

September, 1898. — Loss of strength, dyspnœa on exertion, swelling of abdomen, liver border below umbilicus.

May, 1900. — Confined to house, feet begin to swell, abdomen continues to enlarge.

August, 1900. — Slight amount of blood per rectum.

November, 1900. — Emaciated, icteric, liver border as in photo.

May, 1901. — Very weak, emaciated, ascites, marked hydrops of legs.

August, 1901. — Death. Known duration, 35 months.

Post-mortem examination, August 20, 1901.— B. C. H., U.01.24.— Body of a gracefully built, emaciated man 166 cm. long. Rigor mortis present. Slight yellowish tinge to the skin. Marked œdema of legs, the skin of which is tense and shiny. Abdomen is immensely distended, and at the umbilicus measures 111 cm. in circumference. The lower border of the ribs flares out at a wide angle to contain the immense tumor within.

The abdominal wall is very tense and very thin. Through it the nodulation of the tumor is distinctly visible. On opening the abdominal cavity a considerable amount of fluid escapes under pressure. The abdomen contains several thousand cc. of clear fluid. The entire upper and middle portions of the cavity are occupied by a greatly enlarged nodular liver. The point of insertion in the liver of the round and falsiform ligaments is displaced to a point 11.5 cm. to the left of the median line. The distance between the tip of the ensiform cartilage and the lower border of the liver is 25 cm. From tip of the right ninth rib to the lower border of the liver is 22 cm., from the tip of the left ninth rib to the lower border is 20 cm. From the costal margin to the insertion in the liver of the round ligament is 13 cm. The liver is entirely free from adhesions, and is held in place only by the normal ligaments. The diaphragm reaches to the third rib on the right side, to the fourth space on the left side.

The stomach lies behind the tumor mass, about in the normal position, and shows no nodules or other abnormality. Just below the border of the liver a portion of the transverse colon appears. Below this the space is occupied by coils of small intestine. Duodenum, jejunum, ileum, and colon are normal.

Pancreas is normal.

Mesenteric lymph nodes not enlarged. A few of the retroperitoneal lymph nodes in the region of the pancreas are slightly enlarged, white, and firm.

The spleen is crowded into the left upper quardrant of the abdomen, is firmly adherent to the diaphragm, and is not enlarged.

The kidneys occupy a normal position. On section they are pale and firm. The capsule is slightly adherent, tearing away a small amount of kidney substance on removal.

Adrenals are normal.

The right lung is firmly adherent everywhere to the parietes. It is greatly compressed and the lower half is entirely atelectatic. The upper half is air-containing, but very œdematous.

The left lung is free from adhesions, air-containing, and very œdematous. On the surface of the lower lobe of the left lung are numerous whitish and reddish areas 2 to 3 mm. in diameter, composed of many short, delicate, filiform projections.

The heart is normal.

The aorta shows a few small patches of sclerosis.

In the rectum the mass described below can be palpated, but does not occupy a great portion of the pelvis, and there are no evidences of its having caused any obstruction.

Bladder and prostate normal.

The liver (weight, 15,110 gms., 33.31 pounds avoirdupois) measures 38 cm. in greatest length from right to left border, 39 cm. in greatest length from superior to inferior border, and 19 cm. in greatest anteroposterior diameter. It is coarsely nodular. (Plate III.) The nod-

PLATE III.

ules vary in diameter from 2 cm. to 8 or more cm., and project above the general surface from .5 to 3 or 4 cm. With the exception of these coarse nodules the surface of the liver is smooth and glistening, and there are no evidences of adhesions except along the lines of attachment of the normal ligaments of the liver. The general surface of the liver is dark red to pale red or pink in color. It is mottled with numerous small and fairly large, almost white areas, and a smaller number of somewhat similar, but bluish-black areas. Many of the larger nodules are mottled in color, dark red, brown, bluish-black and white. None of the nodules show umbilication, but on palpation some suggest fluctuation, as if the central portions were much softened. The superior surface to the right of the falsiform ligament is almost smooth in contradistinction to the coarsely lobulated appearance of the other parts of the liver, and is mottled with many smal whitish and greenish areas, none of which measure more than .5 cm.

On section (Plate IV.) the liver shows large and medium-sized, rounded areas of a dark red color. About some of these there appears to be an indefinite connective tissue capsule, but many are in direct contact with liver tissue. Between these masses the liver is yellowish-gray and appears fairly normal. There are some areas of liver tissue as large as 5 x 7 cm. None of the nodules seen in section show evidence of central softening.

The gall bladder is normal, free from adhesions, lies in a deep groove between nodular projections of the liver, and is dark green in color. Bile ducts normal.

PLATE IV.

The portion of the inferior vena cava attached to the liver is normal.

In the rectum, situated approximately 7 cm. above the internal sphincter ani muscle, there is a slightly nodular, soft, brownish-black excrescence which projects 1 cm. into the lumen of the rectum. The base of this is formed by a hard, indurated mass, whose maximum diameter is 3 cm. There is no evidence of ulceration of mucosa in this region. In the areola and connective tissue between the rectum and the sacrum is a hard, indurated mass, 10 cm. long, 3 cm. broad, and

nowhere more than 1 cm. thick. This follows up behind the rectum, but is nowhere attached to it except at the site of the above-described excrescence. This excrescence is situated 2.5 cm. above the lower limit of this indurated mass.

Section through the middle of the nodule in the rectum shows an outer irregular border of purplish-black gradually shading into grayish-white in the centre. This tissue is soft. Beneath this the tissue is hard, in places is glistening white, in others reddish with a few narrow bands of glistening white.

Anatomical Diagnoses. — Adeno-carcinoma of rectum, invasion of post-rectal tissues, metastases in post-peritoneal lymph nodes and in liver, compression and atelectasis of right lung, slight chronic diffuse nephritis, anasarca, hydroperitoneum.

Microscopic Examination. — In sections through the primary nodule in the rectum the surface is covered by mucous membrane. The outer one-third of this is necrotic, and shows no nuclear stain. The deeper parts stain fairly well. Much of the epithelium lining the glands has desquamated. That remaining intact is of the type normal to the rectum. In some places the mucosa is sharply limited, and the bases of the glands lie in a supporting framework of fibrous and elastic connective tissue, beneath which there is a well-formed muscularis mucosæ layer. In other places there is no such line of demarcation, and the glands pass directly into the deeper tissues. All evidences of the other layers of the intestine — submucosa, muscularis and serosa — have disappeared. In place of these there is an adenomatous growth, subdivided by numerous connective tissue trabeculæ, and this constitutes the entire thickness of the rectum at this point.

Where the normal glands of the rectum pass into the underlying tumor the structure is that of fairly regular tubular glands, separated by narrow bands of loose connective tissue. This represents the simplest form of the growth. The greater part of the rectal growth and the metastases in the lymph nodes (post-rectal and post-pancreatic) and liver are of a greater complexity.

In the latter the intertubular stroma is greatly reduced, often being represented by a single strand of connective tissue or a slight meshwork in which a small blood vessel is supported. The tubules have elongated and subdivided. The lumen in places is dilated, and filled with desquamated cells or red blood globules; in others it is narrowed, or has entirely disappeared by coalescence of the two layers. The result of such changes is the formation of large cellular areas separated by broad dense connective tissue trabeculæ. In the cellular areas there is a very small amount of loose connective tissue in which the cells are grouped into gland spaces. Everywhere this adenomatous structure is present, though often obscured by desquamation of cells or approximation of two layers of lining epithelium. It is very evident where hemorrhage has taken place into the lumina, and distended them into definite spaces lined by epithelium.

The epithelial cells of the tumor are columnar. Their height varies but in a given field, they are of quite uniform size. They have oval vesicular nuclei, with fine chromatin granules. Most of them show one or two coarser chromatin granules, though irregular in contour, sug-

gesting nucleoli. The nuclei are situated distally or in the mid zone of the cell rather than at its basal end. Mitotic figures are not numerous. The cytoplasm is homogeneous. Nowhere are there goblet cells. The epithelial cells form a layer, in places one cell deep, in others several cells deep. The desquamated cells are round, and their nuclei are often shrunken and stain intensely. In many places the epithelial layer has separated from the basal membrane. The stroma of the tumor consists almost entirely of fibrous tissue, but in the coarser trabeculæ there are numerous elastic fibres without any apparent relation to blood vessels, beside those in the walls of the blood vessels.

The post-peritoneal lymph nodes show metastases of the same character as the primary growth in the rectum. In some nodes this almost completely replaces lymphoid tissue; in others it is present as very small nodules.

In the liver the growth is similar, though here there has been very extensive hæmorrhage into the nodules. In most of these areas the red blood globules are discrete and remarkably well preserved. The liver tissue about these nodules is compressed, and the liver cells much atrophied. Radiating out between the liver lobules are broad bands of connective tissue, often containing new-formed bile ducts. There is a slight intralobular connective tissue increase, and in places small groups of separated atrophied liver cells are enclosed in the connective tissue trabeculæ. There is very little evidence of necrotic changes in these liver nodules.

The villous processes described on the pleural surface of the lung are composed of loose vascular connective tissue. The other organs present no noteworthy lesions.

The greatest interest of this case lies in the enormous size of the liver. That secondary carcinoma produces the largest livers is generally conceded, and numerous cases have been reported in which the liver attained great size. Thus there are a number of cases with weights between 8,000 and 10,000 gms. (18 to 22 pounds, avoirdupois). Gordon[1] describes one of 24 pounds and Axel-Key[2] one of 25 pounds. Gould and Pyle[3] ascribe to Kraus a case in which the liver weighed 25 pounds and to Gooch one weighing 28 pounds. We have found one case exceeding ours in weight. This is one reported by Powell.[4] He says, speaking of the size of the liver: "At a medium its ordinary weight may be estimated at about 3 pounds or somewhat more; but the variations

[1] Gordon. A Case of Cancer of the Liver; Rupture of its serous covering; compressed Heart. Dublin Quart. Jour., Nov., 1867.

[3] Axel-Key. Hygiea, 1865, xxvii., No. 5. Cited by Leichtenstern.

[2] Gould and Pyle. Anomalies and Curiosities of Medicine, Phila., 1897.

[4] Powell. Observations on Bile and its Diseases; on the economy of Liver. Goulstonian Lecture, 1799. 8° Lond. 1800.

of its weight and relative bulk are in many cases of disease most striking. I have seen a liver weighing a little short of 40 pounds." However the nature of the process in these last 3 cases is not stated.

A second point of interest in this case is its long duration, more than 35 months. Most writers state that a duration of 1 year is unusual, and those cases lasting 2 years are very exceptional. Leichtenstern [1] in 19 cases, collected from the Tübingen clinic and statistics of Frerichs and Murchison, finds an average duration of 20 weeks. In 25 cases, from Biermer's clinic, the average is 17 weeks. In 13 of our cases, in which the approximate duration could be estimated, the average is 25 weeks.

It is in just such a case as the one we have reported that a long duration might be expected. The primary growth was in the rectum, and offered no obstruction to the lumen of the intestine. Hence interference with the digestive functions was reduced to a minimum. With a good appetite, and no intestinal obstruction, nutrition was maintained, and in this case the symptoms seemed to depend on the mechanical obstruction of the enlarged liver. On the other hand the larger number of cases follow a primary growth in the upper gastro-enteric tract, where there is greater possibility of hinderance to metabolism and the downward course might be expected to be more rapid. Again with the longer duration there was greater chance for liver enlargement. It is of interest to note in this connection that the second largest liver in our series also occurred in a case where the primary growth was low down in the gastro-enteric tract, but here unfortunately the patient was admitted in a moribund condition, and no history was obtainable.

In conclusion we wish to express our thanks to Dr. Clement and to the clinical and pathological staffs of the Boston City Hospital for data and assistance.

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[1] Loc. cit.

Description of Plates.

Plate I. Liver weighing 8,100 gms. in situ. Adeno-carcinoma secondary to a growth in the sigmoid flexure of the colon.

Plate II. Patient nine months prior to death. Lines marked on abdomen indicate lower border of liver as made out by palpation. Liver in this case at death weighed 15,110 gms. Showed involvement by adeno-carcinoma secondary to a growth in the rectum.

Plate III. Inferior surface of same liver.

Plate IV. Cut surface of same liver.

XV.

SUBPHRENIC ABSCESS AS A COMPLICATION OF APPENDICITIS.

HENRY A. CHRISTIAN, A.M., M.D., AND LOUIS C. LEHR, M.D., OF BALTIMORE.

IN a study of 4,028 autopsies collected from the following sources,

The Boston City Hospital, Boston, Mass., Jan. 1, 1896–Aug. 14, 1902	1,890
The Johns Hopkins Hospital, Baltimore, Md., May 28, 1889–Aug. 13, 1902	1,978
The Rhode Island Hospital, Providence, R. I., July 1, 1900–July 1, 1902	160
	4,028

death was found to be due directly or indirectly to acute appendicitis in 86. Of these 86 cases, 7, or 8.13 per cent., showed an involvement of the subphrenic region in a purulent process, and though in a strict sense of the word not each one of these was a case of subphrenic abscess, yet a review of them may be of use in illustrating the manner of formation of subphrenic abscess secondary to appendicitis, and their comparative frequence.

Since the classical publication of v. Leyden (Ueber Pyopneumothorax subphrenicus, Zeitschr. für klin. Med., I., 1880, 320.), much interest has been attached to the subject of subphrenic abscesses in their various forms. They may occur in either subphrenic space. If in the right, they will be bounded above by the diaphragm, below by the liver, to the left by the suspensory ligament of the liver (Ligamentum

suspensorium hepatis) and elsewhere by the abdominal parietes. If they occupy the left subphrenic space, to these boundaries will be added below, the stomach and spleen, but this space is not so sharply circumscribed as the right. Between the two, the suspensory ligament of the liver forms a barrier, which prevents, as a rule, the abscess from being bilateral. However, the ligament may be eroded and the process extend from one side to the other. Very exceptionally the process has extended from one side to the other behind the liver in front of the vertebral column, or abscesses have originated simultaneously on the two sides, so that the entire subphrenic space is occupied by purulent material, but separated into two portions by the intact suspensory ligament.

The origin of these abscesses is very various. Those on the right side more commonly arise by direct extension of an inflammatory process from the liver, from appendicular and peri-appendicular lesions, from perforation of the duodenum, and more rarely of the stomach; on the left side perforation of the stomach is the most frequent cause. Of the total number of cases, the cause, according to Nothnagel (Spec. Path. u. Therapie, Wien, 1898, XVII., 718), lies perhaps most often in a round ulcer of the stomach. Other etiological factors are inflammatory conditions of the kidneys, gall bladder and ducts, echinococcus of the liver, and extension of an inflammatory process from the thoracic wall or contents. These abscesses may contain only purulent material (abscessus subphrenicus) or purulent material mixed with gas (pyopneumothorax subphrenicus of v. Leyden). The gas comes either from perforation of some air containing viscus, or is produced *in situ* by the bacterial flora present, either the bacillus ærogenes capsulatus (Welch) or some one of the bacillus coli communis group. This last form may occur as a complication of appendicitis, as in a case reported by Umber (Pyopneumothorax subphrenicus (Leyden), auf perityphlitischer Basis ohne Perforation, Mittheil. a. d. Grenzgeb. d. Med. u. Chir., VI., 1900, 605). These two forms are often separated, since the latter presents a very distinct clinical picture, but etiologically and anatomically they are the same.

Subphrenic abscess is not a very common disease. Maydl in his monograph (Ueber subphrenische Abscesse, Wien, 1894) collected 179 cases. Lang (Ueber subphrenische Abscesse, Diss., Moscow, ref. Jahresbericht der gesammt. Med., II., 1895, 353) collected 173, and added three new cases. Yet, judging from its frequency following appendicitis, subphrenic abscess is a much more common affection than the above figures seem to indicate. Furthermore, during the past two years, among 410 autopsies at the Boston City Hospital, there have been six cases of subphrenic abscess, exclusive of those following appendicitis. Of these, three were right-sided and three were left. The former followed a perforated gastric ulcer, acute suppurative cholecystitis, and typhoid respectively; the latter pyothorax, cholelithiasis, with suppurative cholangitis but no liver abscess, and acute hæmorrhagic pancreatitis. In two of the cases the diaphragm showed gross perforation.

Of Maydl's 179 cases, 23 were secondary to appendicitis. Lang found 26 due to appendicitis among 176 cases of right-sided subphrenic disease. Sachs (Archiv. f. klin. Chir., I., 16) reported 41 cases in which he included 25 of Madyl's cases, but two of these should have been excluded, because the primary source lay in a perforation of the cœcum by a foreign body. Weber (Deutsch. Zeitsch. f. Chir., LIV., 1900, 423) reports 9 cases from 600 cases of appendicitis operated on by Sonnenburg. Of these 600 cases, 350 had a peri-appendicular abscess, so the 9 cases of subphrenic abscess form 2½ per cent. of the abscess cases. To these, in a later paper, Weber (Deutsch. Zeitsch. f. Chir. LX., 1901, 127) has added 5 cases from Sonnenburg's clinic. Elsberg (Annals of Surgery, XXXIV., 1901, 729) has operated on two cases of subphrenic abscess among 91 cases of appendicular disease, and from medical literature has been able to collect 71 cases of subphrenic abscess complicating appendicitis, making, with his two, a total of 73 cases. In doing this he excluded a number of cases, because the reports were meagre and incomplete. Since the date of his publication single cases have been reported by Blake (Annals of Surgery, XXXIV., 1901, 703), Gaston (Med. Record, LIX., 1901, 452), Robinson

(Lancet, 1899, I., 209), Dale (Cincinnati Lancet-Clinic, XLVI., 1901, 451), Caley (Lancet, 1900, I., 378), and others.

With Weber's 2½ per cent. of 350 operated abscess cases, Elsberg's 2 cases in 91 consecutive appendicitis operations, and the frequency with which we have found at autopsy an inflammatory condition in the subphrenic region, the conclusion seems justified that subphrenic abscess is not a very unusual complication of appendicitis, but that these cases have not, as a rule, been reported.

As these seven cases present a number of differences and several points of interest, those parts of the autopsy protocols relating to the appendix and the peritoneal and pleural cavities will be given in some detail.

Case 1. — B. C. H., 98.247. J. H., male, age twenty-six.

Anatomical diagnoses. — Scar of former appendix operation; acute fibrinous general peritonitis; chronic adhesive localized peritonitis; retrocœcal, perisplenic and subphrenic abscesses; right pyothorax; acute serofibrinous pleuritis, left side; complete atelectasis, right lung; partial atelectasis, left lung; acute bronchitis; acute purulent mediastinitis; volvulus of small intestine; thrombosis of vessels of liver.

In the right groin is a scar 4½ cm. in length running parallel to Poupart's ligament, which is dark bluish in color, and slightly depressed below the surrounding skin (operation six months ago for appendicitis).

Peritoneal cavity. — On opening the abdomen the coils of the small intestines present are very greatly distended, hyperæmic, and covered with thin flakes of fibrin. The coils of intestine are united by easily broken fibrinous adhesions. In the region of the appendix and cœcum are numerous firm fibrous adhesions, in the meshes of which the appendix, with obliterated lumen, is found. The diameter of the appendix is much decreased. Posterior to the cœcum is found an abscess cavity, containing a considerable amount of thick, creamy pus. The spleen is walled off from the general cavity by firm adhesions, and lies in a mass of the same thick, creamy pus. This pus is very adherent to the capsule of the spleen, and is only scraped off with some difficulty. The right lobe of the liver is bound to the diaphragm by recent adhesions. On separating these there is found to be a layer of the same thick, creamy pus lying between the liver and the diaphragm on the right side. Immediately over the left lobe of the liver there are fresh adhesions, but no pus. On examining the small intestine, 120 cm. above the ileocœcal valve a complete twist of the intestine is found. All of the small intestine below this point and the large intestine is collapsed.

Pleural cavities. — The right pleural cavity contains 1800 cc. of thick, creamy pus. The pleura is thickened and covered with soft yellowish fibrin flakes. The left pleural cavity contains 800 cc. of a dark reddish

fluid in which are numerous large flakes of fibrin. The fluid here is not purulent. The parietal pleura is also adherent, shows the same appearance as the right, and in addition there are numerous small hæmorrhages beneath it.

Lungs. — Right lung occupies a position close to the spinal column, is flattened, adherent by its apex to upper chest wall, slate-colored, and covered by numerous thick yellowish soft fibrin flakes. The lung is exceedingly heavy and flabby. On section it cuts with difficulty, is completely airless, and can be readily moulded into almost any shape. Bronchi injected, and contain considerable muco-pus.

Left lung shows the same condition of its pleura, and along the backs of the upper and middle lobe is the same condition as in the right lung, but the inner and anterior portions of the lung still contain air, are rather soft, pinkish yellow in color, and cushiony. Bronchial glands are enlarged and pigmented.

Diaphragm. — Both sides covered with an exudate of fibrin. No perforation present.

Cultures. Heart's blood — Streptococcus pyogenes.

Spleen. — Streptococcus pyogenes, Micrococcus lanceolatus, and Bacillus coli communis.

Kidney. — Sterile.

Right pleural cavity. — Streptococcus pyogenes, Micrococcus lanceolatus.

Subphrenic abscess. — Streptococcus pyogenes in almost pure culture.

Case 2. — B. C. H., 00.115. E. A., female, age thirty-three.

Anatomical diagnoses. — Peri-appendicular, pelvic, and subphrenic abscesses; chronic localized adhesive peritonitis; acute fibrino-purulent pleuritis, right side; atelectasis, right lung; operation wounds.

In the right iliac region there is a healed scar 7 cm. long. In this same region, external to above scar, there is an incision 7 cm. long which enters the peritoneal cavity at the right side of the cœcum, and through it a gauze drain passes into the region of the appendix.

Peritoneal cavity. — The surface of the general peritoneum is smooth, glistening and dry. The region of the appendix and the end of the cœcum are surrounded by slight adhesions and bloody diffluent material. The point of origin of the appendix is difficult to find, and its stump is apparently perfectly healed. The pelvis is walled off by rather strong adhesions involving omentum, small intestine, sigmoid flexure and apex of the bladder. On separating these the pelvis is found to be filled with dirty pus-like material. No direct connection between this and the condition about the appendix was to be made out. Between the diaphragm and the right lobe of the liver there is a small amount of dirty pus-like material. This communicates with the exterior by means of a wound passing through the base of the right pleural cavity.

Pleural cavities. — The left pleural cavity is normal. The anterior part of the right pleural cavity is also normal. The lateral and posterior parts of the right cavity from base to apex and the right half of the base are walled off by rather firm fibrinous adhesions, and filled with 700 cc. of fibrino-purulent exudate. The pleural walls are here covered by thick layers of fibrin. This cavity communicates with the exterior by above-mentioned incision.

Lungs. — Left lung pink, downy and appears normal. Right lung in the posterior and apical portions is normal. In the other portions it is covered with fibrinous exudate. Its tissue is soft and spleen-like in appearance. A bit of this sinks in water.

Cultures. Heart, Spleen, Liver, Kidneys, Lungs, Pleuræ and Peritoneum — Bacillus coli communis.

Case 3. — B. C. H. 01.110–H. B., male, age thirty-five. Anatomical Diagnoses. Operation wounds; appendectomy; acute fibrinous general peritonitis; mesenteric and portal pyophlebitis; multiple abscesses of liver; acute splenic tumor.

Skin generally has a slight icteric tint and conjunctivæ are distinctly yellow. In the median line is an incision through the abdominal wall 8 cm. in length, beginning 1 cm. above the umbilicus and extending to within 3 cm. of xyphoid cartilage. In the right lower quadrant of the abdomen is an "appendix" incision 4 cm. in length. Both wounds are closed by sutures.

Peritoneal cavity. Peritoneal surface is lustreless. All the coils of bowel are slightly adherent by easily broken fibrinous shreds. In the cavity of the pelvis is a small amount of sero-sanguinous fluid. In the region of the appendix the appendix stump is found, and on pressure nothing escapes from the lumen of the cœcum. Between the spleen, stomach, left side of diaphragm, liver and posterior wall of peritoneal cavity is an abscess containing offensive, yellowish, semi-fluid, purulent material. Its wall is everywhere covered by a fibrinous membrane, about 1 cm. in thickness, which is readily detached. Smears from pus in the subphrenic abscess show flattened streptococci. Mesenteric lymph nodes all somewhat enlarged.

Pleural cavity.—Left pleural cavity is normal. Right pleural cavity is obliterated by old fibrous adhesions throughout. No fluid.

Lungs. — Left lung normal in color and consistence. Surface of the right lung is beset with fibrous shreds. On section it is normal.

Cultures. Heart's Blood. — Streptococcus pyogenes, 1 culture; Bacillus coli communis, 3 cultures; Sterile, 1 culture.

Liver. — Streptococcus pyogenes and Bacillus coli communis, 2 cultures.

Spleen. — Bacillus proteus, 1 culture.

Kidney. — Bacillus coli communis, 1 culture.

Peritoneum beneath omentum. — Sterile, 1 culture.

Blood from vessel near Portal vein. — Streptococcus pyogenes, 1 culture; Bacillis coli communis, 1 culture.

N. B. — Cultures made from heart and blood vessels by aspirating with syringe and adding blood to media.

Case 4. — B. C. H., 02.91-G. C., male, age 33. Anatomical diagnoses. Operation wound; appendectomy; perityphlitis; volvulous of small intestine; acute serofibrinous peritonitis; subphrenic abscesses; chronic tuberculosis of lung and spleen; tuberculous ulcerations of ileum, chronic interstitial nephritis.

Midway between umbilicus and anterior superior iliac spine there is an operation wound running parallel with the rectus abdominis muscle.

Peritoneal cavity. There is about 200 cc. of greenish yellow fluid in the subdiaphragmatic regions of both sides. Smears made from this show a variety of bacteria including the streptococcus pyogenes. The coils of intestine are markedly meteoristic. The exudate in the lower abdomen is less fluid and more fibrinous than higher up, and glues together the coils of the intestine. The subdiaphragmatic surface of the liver shows fresh fibrin in smooth flakes, which can be easily peeled away. The right lower angle of the liver shows greenish fibrin and an exudate which appears to have affected the liver substance itself.

Two to three cm. internal to the appendix incision, a portion of the wall of the cœcum has become firmly adherent to the abdominal wall and presents a fibrous ragged surface on separation with considerable old hæmorrhage. The appendix can not be found (at operation a gangrenous appendix already sloughed off had been removed). Its stump is buried in a blackish mass of tissue lining the inside of the deep pocket beneath the appendix incision.

Pleural cavities. Left normal. Right completely obliterated by old adhesions.

Lungs normal, except for a caseous nodule in middle lobe of right lung.

Case 5. — R. I. H., 00.1. J. C., male, age fifty. Anatomical diagnoses. Operation wound; acute ulcerative appendicitis; peri-appendicular abscess; extension of abscess into retroperitoneal and perinephric tissues; perforation of ascending colon; acute localized peritonitis; œdema of lungs.

There is an operation wound in the right groin, beginning 2 cm. to inner side of anterior superior spine of the right iliac bone, and extending upwards and outwards parallel to the crest of the ilium for a distance of 6 cm. Abdomen is distended.

Peritoneal cavity.— Peritoneum is smooth, except about seat of operation. The mesenteric lymph nodes are not enlarged. No fluid in peritoneal cavity. Intestines are distended with gas. There are slight fibrinous adhesions of intestines to abdominal wall around operation wound. These adhesions are easily separated. The separation exposes two openings — one just external, the other internal, to the cœcum, both extending into the retroperitoneal tissues. From them a yellowish semi-fluid fecal material escapes.

Appendix. — only the base is visible. It arises from the posterior wall of the lower end of the cœcum, and extends inward along the brim of the pelvis, with the tip slightly curled upon itself. It is firmly bound down by old fibrous adhesions. It measures 12 cm. in length. The proximal 6 cm. appear perfectly normal. The distal 6 cm. is riddled by multiple perforations, from some of which escapes a yellowish fecal fluid. The abscess about these perforations is found to communicate with the retroperitoneal space, into which the above-mentioned openings about the cœcum lead. In the posterior wall of the coecum is an almost spherical opening about 1½ cm. in diameter leading into the retroperitoneal tissues also. The retroperitoneal tissue here is softened and diffusely infiltrated with a brownish-gray puriform material. This extends upwards as a grayish-

yellow exudate in places muco-purulent, elsewhere as masses of a tough, apparently fibrinous material. It invades the right psoas muscle, extends along the vena cava inferior, posterior to the duodenum as it crosses the spinal column, and infiltrates everywhere the perinephric tissues of the right side, extending even between the diaphragm and the liver.

Pleural cavities. — Left is smooth; right is almost entirely obliterated by old fibrous adhesions.

Lungs are deeply pigmented. Left is very voluminous, especially the lower lobe, but is soft and crepitant throughout. It is very dark red, and on section presents a smooth, moist, glistening surface, from which much bloody fluid escapes on pressure. Right is not quite so voluminous as left, but otherwise not essentially different. Bronchial lymph nodes pigmented, but not enlarged.

Cultures. Heart's blood. — Streptococcus pyogenes.

Peritoneum. — Streptococcus pyogenes.

Perinephric tissue. — Streptococcus pyogenes.

Bits of tissue from liver, lung, heart and kidney, placed in thermostat over night, all show large numbers of bacilli, with the morphology and staining properties of the bacillus ærogenes capsulatus.

Case 6. — J. H. H., 1581. Female, age thirty-two. Anatomical diagnoses. — Operation wounds; acute gangrenous appendicitis; periappendicular pelvic abscess; subphrenic abscess; acute fibrino-purulent general peritonitis; acute splenic tumor; cloudy swelling of myocardium, kidneys and liver; œdema and congestion of lungs; healed tubercular foci in lungs; acute sero-fibrinous pleuritis, both sides; chronic adhesive pleuritis, both sides.

Under the right edge of rectus abdominis muscle is a recent surgical incision 17 cm. in length, with the middle third containing a gauze drain and the upper and lower thirds closed and healing per primam. In the right flank postero-laterally is a recent surgical incision 3 cm. in length, containing drains.

Peritoneal cavity. — Upon opening the peritoneal cavity the intestines are found adherent to the anterior abdominal wall by firm fibrinous adhesions, which are found everywhere except in the region of the splenic flexure of the colon. Between the loops are numerous pockets of pus. Upon untwisting the coils of intestine, it is found that the appendix is gangrenous, and has sloughed off at its attachment to the cœcum. The detached portion dips over the brim of the pelvis, and is contained in a large abscess containing about half a pint of thick grayish-yellow, foul-smelling pus. The abscess cavity occupies Douglas' cul-de-sac. The stump of the appendix is shut off by one of the gauze drains. The other gauze drain lies against the colon on its lateral side. A rubber drain leads into an abscess cavity between the liver, hepatic flexure and lateral abdominal wall. The liver has between its right lobe and the chest wall an extensive abscess which extends also over the lower surface of the diaphragm almost to the median line.

Pleural cavities. — The right plural cavity contains 100 cc. of turbid fluid, containing flakes of fibrin. Left pleural cavity contains about 200 cc.

Lungs. — Left lung roughened over lateral and posterior surfaces from old adhesions, studded with ecchymoses and moderately voluminous. The dependent portions not crepitant. At the apex of the lower lobe are several firm nodules, which on section are found to be caseous and calcifying. On section the lung is found to contain a great excess of watery, frothy fluid. The lung has a moderately uniform color, from a dark red in the lower lobe to a higher red in the upper lobe. There are one or two emphysematous areas in the lower lobe.

Right lung. — Moderately voluminous. Pleural surfaces smooth, except for a few old adhesions at the base. At apex is a puckered scar containing a calcifying tubercle. The dependent portions of the lung are not crepitant. On section the lung contains an excess of a frothy, watery fluid, and presents the features seen in the left lung. The non-crepitant lower lobe sinks in water.

Case 7. — J. H. H., 1865. Male, age eleven. Anatomical diagnoses. Appendectomy ; acute general fibrinous peritonitis ; extensive multiple abscess formation in the peritoneum ; subphrenic abscess ; acute diphtheritic colitis ; acute pleuritis, left side.

Peritoneal cavity.—Upon opening the peritoneal cavity there are found delicate fibrinous adhesions everywhere between the abdominal organs and the anterior abdominal wall. There is a small abscess in the subcutaneous tissue communicating with the granulating surface of the wound by a channel at the lower end. The abdominal organs are very much bound together, and on attempting to separate them one ruptures the intestines. There are delicate adhesions between intestinal coils everywhere. Just under the diaphragm on the left side is a large abscess cavity located outside of the stomach, partially walled off by the liver. This contains greenish-yellow pus. This cavity is lined by a rough hyperæmic granulation tissue which is thrown up into folds which are almost like the valvulæ of the intestines. Pressure on the abscess causes the pus to exude from another cavity in the ileo-cæcal region, and a probe can be passed 30 cm. without meeting with resistance along the right abdominal wall. The channel can be traced with the scissors from the subphrenic abscess into the ileo-cæcal region, where it turns toward the centre, giving rise to numerous channels which pass down between the intestinal coils. Another abscess filled with fluid is found just behind the transverse colon ; this cavity is continued around over the aorta, where it overlies the ascending colon. The ileo-cæcal valve is found to be practically in its normal position. In the walls of the abscess there are black silk sutures at the point where the cœcum is against the abscess cavity ; this is probably the seat of the appendix. On the left side midway down the course of the abscess cavity there is a fistulous communication with the ileum. The kidney lies quite free behind the abscess. The transverse colon at the splenic flexure crosses the abscess and lies behind it ; the abscess is not therefore retroperitoneal. The channel follows almost exactly the course of the colon down to the region of the bladder, where it crosses over through the sinus, which opens on the abdominal surface. The abscess cavity on the right side extends up to the region of the liver, where it ends blindly. The pancreas is free and apparently normal. The main

coils are inextricably embedded together, and there are numerous adhesions and abscesses between these coils. An attempt to separate them results in tearing the intestines. The abscesses lie very often between the adhesions of the mesentary and omental folds. Adhesions are more dense about the region of the ileo-cæcal valve.

Lungs. — Left lung is somewhat collapsed in the lower lobe, where it is firmly adherent to the diaphragm.

Of these cases, 5 were male, 2 female. One was 11 years old, one 26, 4 between 30 and 35, and one 50. In 4 of these cases the streptococcus pyogenes was present in several organs, in one the bacillus coli communis, and in two no cultures were taken. In 5 cases the appendix had been removed, leaving only a short stump; in one the cœcum and the appendix with obliterated lumen were bound down by fibrous adhesions, and behind the cœcum was an abscess; in one the appendix was bound down by adhesions, its distal half gangrenous and riddled with perforations.

In the above 7 cases the affection was unilateral 6 times, 4 times on the right, 2 times on the left. In one case it was bilateral. Right-sided subphrenic abscess is much the more common following appendicitis. The cases of Lang, Weber, Elsberg, Blake and Umber were all right-sided. Strangely enough, Elsberg (*loc. cit.*) does not seem to consider the left subphrenic region in treating the subject, and in his table of collected cases the locus of the disease is not mentioned. If we consider the way in which the abscesses originate secondary to disease of the vermiform appendix it is evident that the right-sided form would be the most common, though the left can readily occur, the localization being somewhat influenced by the situation of the appendix.

Subphrenic abscess secondary to appendicitis may occur in one of four ways:

(1.) As a localized abscess, a part of a general purulent peritonitis.

(2.) By extension of the diseased process from the appendix to the subphrenic region by an intraperitoneal route.

(3.) By extension of the diseased process by an extraperitoneal route, either by way of the lymphatics or by infiltration through the retroperitoneal tissues.

(4.) By way of the blood current as part of a general embolic septic process, or as a sequence of liver abscesses which are of embolic origin by way of the portal vein.

Originating in either the first or last ways the abscess might equally well appear on either side; in the second, either side might be affected, but the right would be much more frequently, since the ascending colon forms a natural drain or gutter in this direction; in the third only rarely would the left side be involved, and then, as a rule, accompanying a misplaced diseased appendix.

It is by extension (2) and (3) that the greatest number of these cases originate. Whether the route in any given case will be intra- or extra-peritoneal depends on the situation of the appendix and the peri-appendicular process. If the latter is retrocœcal, and so extraperitoneal, the chances are that the process will take an extraperitoneal route and reach the subphrenic region by way of the loose areolar tissue in the lumbar region. When the abscess is retrocœcal, it is claimed that there is greater liability to the formation of a subphrenic abscess.

Having reached the subphrenic region extraperitoneally, the abscess may remain so or it may then become intraperitoneal. If the former is the case it is likely to be small, since the peritoneum covering the diaphragm is very intimately adherent to the muscle substance (Hofmann, A. Schmidt, cited by Elsberg). The latter fact would also influence its early penetration into the space between the diaphragm and liver. Among the cases collected by Elsberg 27 per cent. were extraperitoneal, 48 per cent. intraperitoneal and 25 per cent. of doubtful anatomical location.

Our cases illustrate quite well some of these points in the manner of origin of subphrenic abscesses. In Case I. there is a retrocœcal and a right-sided subphrenic abscess. Here it would seem that the latter originated by the extraperitoneal route, though the case is complicated by a volvulus of the small intestine, general fibrinous peritonitis, fibrinous adhesions between the left lobe of liver and the diaphragm and thrombosis of some of the veins in the liver. Case II. also indicates an extraperitoneal route to the right subphrenic

space; here the general peritoneal cavity is clear. In Case III. pylephlebitis and multiple liver abscesses are present. The subphrenic abscess is on the left. Case IV. shows bilateral, not completely walled-off subphrenic collections of pus, part of a general peritonitis. In Case V. an infiltrating purulent process can be clearly traced from the appendix region up along the retroperitoneal tissues of the right lumbar region, until it makes its appearance between the diaphragm and liver. In Case VI. the course of the process is not very clearly traced, though with a general peritonitis and numerous pus-pockets it may have arisen as part of the general peritonitis. Case VII. shows an intraperitoneal route of extension from appendix to left subphrenic region. Among the seven cases it may be claimed that all four ways of origin are illustrated.

In four of the seven cases the process had extended from the subphrenic region into the pleural cavity, though in none was the diaphragm perforated. In Case II. the pleura had been incised by the surgeon for drainage. In the other three the pleural cavity on the side of the subphrenic abscess was entirely obliterated by fibrous adhesions.

In cases of appendicitis the pleura may become involved in an inflammatory process in two ways: (1) by extension from a pneumonic focus or infarct in the lung; and (2) by extension from the abdominal cavity, either by way of the lymphatics or by erosion of the diaphragm. This latter form constitutes the appendicular pleurisy of the French writers, is very often associated with subphrenic abscess as in four of our seven cases, and is almost always right-sided. That it is always right-sided, as claimed by Lapeyre (Revue de Chir., XXIII., 1901, 508 and 646) and others cannot be agreed to; for, as we have seen, subphrenic abscess following appendicitis may occur on the left side, and be followed by acute pleuritis of that side. This did happen in Case VII.

In conclusion, we wish to express our thanks to the pathological departments of the Boston City, Johns Hopkins and Rhode Island Hospitals for access to their records and permission to report these cases.

XVI.

SOME CASES WHICH WERE NOT PERNICIOUS ANÆMIA.

By John Lovett Morse, A.M., M.D.

These cases are reported with the object of emphasizing the writer's belief that the diagnosis of pernicious anæmia cannot be made with certainty on the basis of the condition of the blood alone. Due consideration of the physical signs and of the history of the disease is also necessary. Without them a positive diagnosis is impossible. There is no question, of course, but that the condition of the blood in pernicious anæmia is most characteristic. Identically the same condition may be found in the blood, however, in the course of other diseases. It is these severe secondary anæmias which are mistaken for pernicious anæmia, the diagnosis being made on the condition of the blood without sufficient consideration of the history or careful search for a possible cause.

Case I. — Frans E., twenty-seven, single, a carpenter, was admitted to the hospital October 21. His mother died of phthisis. He drank considerable beer, and had had gonorrhœa, syphilis and rheumatism. He used tobacco, tea and coffee.

Two weeks before entrance he had a chill, which was followed by headache, dizziness, bad taste in the mouth and loss of appetite. Shortness of breath soon developed and steadily increased. There was also a dry cough. He had had several slight chills since the first one. There had been no nose-bleed. The bowels were constipated.

He was well developed and nourished. There was marked pallor of the skin and mucous membranes. The tongue was pale, indented, and covered with a moist, white coat. The right border of the heart was two fingers' breadth to the right of the right edge of the sternum, the left border two fingers' breadth to the left of the nipple line. The

impulse was felt best in the fifth space outside the nipple line. The action was regular. A systolic murmur was heard over the whole præcordia, whence it was transmitted into the axilla and upward into the vessels of the neck. The pulse was of fair volume and tension. There was marked pulsation of the cervical vessels. The lungs showed nothing abnormal except occasional dry and moist râles at both bases behind. The lower border of the liver was palpable. The splenic dulness began at the seventh rib; the edge was palpable. There was no œdema. The temperature ranged from 102° F. to 100° F. The urine at entrance showed nothing abnormal. The blood, examined at entrance, showed 1,216,000 red corpuscles and 4,200 white corpuscles. Pernicious anæmia was suspected. He continued delirious and stupid. There was no improvement under arsenic and iron.

The blood was reëxamined on October 24. The count was essentially the same as at entrance. The stained specimens showed marked variation in the size of the red corpuscles, the general tendency being toward increase in size. There was also a decided tendency toward oval forms. There was slight polychromatophilia and poikilocytosis. Eleven nucleated red cells were seen while counting 200 white cells; one of these was a normoblast, the remainder megaloblasts. One megaloblast showed a nuclear figure. The differential count of the white corpuscles was as follows:

Small mononuclear	30 per cent.
Large "	6 "
Polynuclear neutrophiles	57 "
Eosinophiles	1 "
Myelocytes	6 "

This condition of the blood coincides in almost every way with that found in pernicious anæmia. The low red count, the hypoleucocytosis, the increase in size of the red corpuscles, the tendency to oval forms, the excess of megaloblasts, the increased proportion of mononuclear cells and the considerable percentage of myelocytes are all most characteristic. On the strength of this blood picture the diagnosis of pernicious anæmia was made. The sudden onset, the very rapid deterioration of the blood and of the general condition, although inconsistent with the usual course of pernicious anæmia, were disregarded. They should have suggested a further search for the cause.

This cause was discovered by accident the next day in a more thorough examination of the stained blood, which showed an occasional cresentic form of the æstivo-autumnal parasite of malaria.

Quinine was given in doses of ten grains t. i. d., which were later increased to 15 grains t. i. d. He continued delirious and stupid until about the first of November. He then began to improve a little, and on November 6 sat up in bed, but complained of some vertigo and blurred vision. The temperature had ranged between 99° F. and 100° F. up to this time. It was normal after November 10. On November 11 he sat up in a chair. He was still very weak and still suffered from blurred vision.

The blood on November 14 showed:

Red corpuscles	1,788,000
White corpuscles	6,200
Hæmoglobin	54 per cent.

He continued to improve in every way. The blood on December 3 showed

Red corpuscles	3,328,000
White corpuscles	6,600
Hæmoglobin	75 per cent.

He was discharged well on December 10.

Case II.—Wm. P., fifty-one, married, a baker, entered the hospital February 13. His family history was good. He had had an attack of rheumatism when thirty-six, lasting for five or six weeks. He was ill with pneumonia in the City Hospital when forty-one. The records unfortunately contain no note as to the condition of his heart or blood at that time. The urine was normal. He began to get very fleshy when about thirty years of age. During the last ten years he had been troubled by dyspnœa on exertion and slight swelling of the feet. During the past five years he had always felt tired and ready to lie down. His digestion had been good. He had not been a drinking man except by spells. He used no tobacco and denied any venereal disease. He said he used to be "of good color for a baker." His sight had been dim for a year.

Nine weeks before entrance he began to grow weaker, and four weeks later he had to give up work. The weakness continued to increase so that for a week before entrance he had not been able to sit up more than three hours a day. Associated with the weakness, especially during the last month, had been dizziness, dyspnœa on exertion and tinnitus. He had lost twenty pounds in the last two months. His appetite was fair, his digestion good, his bowels constipated. His bowels had always been constipated, requiring drugs.

He was well-developed and obese. His skin was pasty. The conjunctivæ and mucous membranes were pale. The tongue was clean and moist. The upper border of cardiac dulness was at the middle of the third rib, the right border 6 cm. to the right of the median line, the left 14 cm. to the left of the median line. The action was regular. The sounds were feeble at the apex, where there was a soft systolic murmur, not transmitted. The second pulmonic sound was accentuated. The pulse was regular, but of poor volume and tension. There was a marked venous hum in the neck. The lungs, liver, spleen, abdomen and extremities showed nothing abnormal. There was no enlargement of the superficial lymph nodes. His temperature ranged between 99° F. and 100° F. The urine showed nothing abnormal.

The examination of the blood showed :

Red corpuscles	2,208,000
White corpuscles	4,400
Hæmoglobin	35 per cent.

The stained specimens showed considerable variation in the staining qualities and size of the red corpuscles. There was a tendency toward large, but none toward oval, forms. Poikilocytosis was slight. One megaloblast was seen in counting 300 whites. The differential count of the white corpuscles was as follows :

Small mononuclear	25 per cent.
Large mononuclear	5 "
Polynuclear neutrophiles	66 "
Eosinophiles	3 "
Myelocytes	1 "

Although the condition of the blood in this case did not correspond in every particular to that of pernicious anæmia, it was, nevertheless, very characteristic. When it was taken in connection with the history and negative physical examination, no doubt whatever was entertained as to the accuracy of the diagnosis of pernicious anæmia which was positively made. Hence no further search for some obscure cause of the anæmia was made.

There was no improvement under treatment, however. After he had been in the hospital about two weeks it was reported that some of his stools were mixed with bright red blood. The history was then elicited that he had had bleeding piles for twenty years or more, and that they had bled much more freely recently. Examination showed both external and internal hemorrhoids. Operation was advised, and he was to be transferred to the surgical side for operation. Unfortunately, however, on his wife's advice, he refused operation and went home.

It is impossible to state positively that the anæmia in this case was secondary to the hemorrhoids because no operation was performed, and hence no opportunity was given to study the case after operation. I personally believe, however, that it was, and that operation would have been followed by a gradual and permanent improvement in the blood. Repeated small hemorrhages over a considerable period of time lead to marked changes in the blood which often resemble those found in pernicious anæmia. These changes seem especially likely to occur as the result of the bleeding from piles. In a number of cases of secondary anæmia which I have seen

from this cause, but in which the blood changes were not quite as marked, there has been a gradual return of the blood to normal after operation. In all cases of severe anæmia repeated small losses of blood should always be considered as a possible cause. The diagnosis of pernicious anæmia, no matter what the condition of the blood, should, therefore, never be made until all possible sources of hemorrhage have been investigated and excluded.

Case III. — Dora Z., fifty-four, married, was admitted to the City Hospital on May 1. The history was very imperfect, as she spoke but little English. She had been pale for five years. For four years she had noticed distension of the abdomen. For something more than two years she had been passing bloody urine from time to time. Micturition was associated with burning and pain. Two months before entrance she went to bed because of weakness. This weakness, palpitation and other symptoms of anæmia had been more marked for two weeks.

She was well developed and poorly nourished. The skin was harsh and there was marked loss of subcutaneous fat. The skin was of an extremely pale lemon color. The mucous membranes were also pale. The upper border of cardiac dulness was at the upper border of the third rib ; the right was 2½ cm. to the right of the median line, the left 13 cm. to the left of the median line. The impulse was in the fifth space, 12½ cm. to the left of the median line. There was a loud systolic murmur over the whole præcordia which was transmitted upward into the vessels of the neck and downward into the axilla. Its maximum intensity was in the third left interspace and at the apex. The second pulmonic sound was accentuated. There were a few râles at both bases. The liver and spleen were normal. There was considerable œdema of the feet and ankles.

The urine was turbid, slightly acid, of a specific gravity of 1,009, and contained one-tenth per cent. albumin. The sediment showed much pus, free and in clumps, many large round and highly refractive cells, numerous fatty renal cells and considerable normal and abnormal blood.

The blood showed :

Red corpuscles	1,280,000
White corpuscles	8,000
Hæmoglobin	8 per cent.

The stained specimens showed marked poikilocytosis, marked macro- and microcytosis and some polychromatophilia. Two normoblasts and two megaloblasts were seen in counting 100 white cells. The differential count of the white corpuscles was as follows :

Small mononuclear	12 per cent.
Large mononuclear	37 "
Polynuclear neutrophiles	48 "
Eosinophiles	3 "

Another examination on May 8 showed:

Red corpuscles	1,060,000
White corpuscles	7,600
Hæmoglobin	10 per cent.

The changes in the red corpuscles were more marked, and two megaloblasts were seen in counting 100 white corpuscles.

The patient was shown at this time at a meeting of the Clinical Club as a case of pernicious anæmia. The long history of hæmaturia, as well as the condition of the urine which pointed to some disease of the pelvis of the kidney or bladder, seem to have been disregarded. The emaciation, which is unusual in pernicious anæmia, might also have suggested that there was some cause for the anæmia. The condition of the blood was, on the whole, pretty characteristic of pernicious anæmia, although the normal white count and the very low percentage of hæmoglobin are somewhat unusual when the number of red corpuscles is so low.

Attention was soon after directed more particularly to the bloody urine. On May 12 the urine was turbid, acid, of a specific gravity of 1,013, and contained a large trace of albumin. The sediment contained considerable pus, free and in clumps, considerable normal and abnormal blood and much squamous epithelium.

On May 19 the urine was even more noticeably bloody. It was red, slightly acid, of a specific gravity of 1,009, and contained $\frac{1}{5}$ per cent. of albumin. The sediment showed much pus, much normal and abnormal blood, some round and bladder cells.

On May 31 the liver was palpable 4 cm. below the costal border. The abdomen contained a little free fluid, as did the pleuræ.

On June 1 the examination of the blood showed:

Red corpuscles	2,080,000
White corpuscles	8,600
Hæmoglobin	15 per cent.

There was marked macrocytosis, microcytosis and poikilocytosis as well as polychromatophilia. No nucleated reds were seen.

On June 6 a rather unsatisfactory cystoscopic examination of the bladder was made by Dr. Higgins. One ulcerated patch was seen. The bladder was treated by injections of gelatine.

Examination of the blood on June 9 showed:

Red corpuscles	2,560,000
White corpuscles	14,200
Hæmoglobin	20 per cent.

She died suddenly on June 9.

During the whole stay in the hospital she had six or seven loose movements daily, and vomited occasionally. At times the stool contained a few clots of blood. The urine continued bloody. The temperature was irregular, ranging from normal to 100° F.

No autopsy was allowed. Several pieces of grayish-white, tough tissue with blood clots were removed through the urethra after death. Microsopical examination by Dr. Mallory showed connective tissue, muscle tissue and in places foci of round cells. Irregular cellular areas surrounded by connective tissue in which medium-sized cell masses were irregularly grouped were also seen. The cells of these masses were epithelial in type, lay closely packed, had round or oval nuclei, and showed frequent mitoses. The anatomical diagnosis was carcinoma.

In this case the anæmia was probably due partly to the continuous loss of blood in the urine and partly to the cachexia from the cancer. After the attention was directed to the bloody urine and other diseases duly considered, pernicious anæmia was at once dropped from the list of probabilities. It is merely another instance to show how necessary it is not to attach too much importance to the blood condition alone to the neglect of other methods of diagnosis.

XVII.

BILATERAL RETINAL DETACHMENTS IN THE NEPHRITIS OF PREGNANCY.

Dr. Allen Greenwood and Dr. Edward R. Williams, Ophthalmic Surgeons to the Boston City Hospital.

Mrs. E. L. C. entered the Boston City Hospital (service of Dr. George Haven) January 6, 1902, with the following history: She is 28 years of age, was born in America, and is a housekeeper. Her menstrual functions have been regular. She has given birth to one child at term with normal labor, and had one miscarriage at two months, ten years ago. She menstruated last some time in July, 1901, and her pregnancy progressed normally up to a few weeks ago, when she began to notice dimness of vision, which has increased up to the present time. January 2, 1902, she began to have severe pain in her back, which continued until the evening of January 5, when she had a severe attack of pain in the epigastrium, followed during the night by several convulsions. Has had no convulsions since last night, but has some headache and epigastric pain.

Examination shows a well developed, somewhat obese woman, with normal heart and lungs. Pulse, 120; temperature, 100. Complains that sight is very dim; pupils dilated, but react to light and accommodation. Face somewhat bloated. Abdomen lax, tympanitic, but not tender. Uterus felt about two inches above the umbilicus. Lower extremities œdematous. Mentally dull, but rational. Uterus enlarged to size of six months pregnancy. Os soft, but not patulous. Urine shows dark color, acid reaction, 1018 specific gravity,

¼ to ½ per cent. albumen. Sediment consists of much blood, many hyaline, fine and coarse granular casts, some squamous epithelium, and few leucocytes.

Treatment. — Catheterized, surrounded by heaters and covered with blankets. Alternate doses every three hours of morph. sulph. gr. ¼, and chloral hydrate gr. XV. Copious amounts of diuretics and diaphoretics were administered. No convulsions since entrance. Force cream of tartar water, Basham's mixture every four hours, ounces ½.

January 9, 1902. Condition the same. Urine shows only trace of albumen and the sediment contains fewer casts. Milk and lime water four ounces every four hours; tincture of digitalis m. 10 every six hours.

January 10. Patient very comfortable. Pulse ranges from 90 to 120; temperature 99 to 100. There have been no convulsions, but patient has been more or less restless at night; has occasionally tried to get out of bed, although her mind has been clear. Complains of blurring and dimness of vision. Urine has steadily increased in amount until to-day, when thirty-seven ounces were passed.

January 14. Patient complained of a great deal of epigastric pain, and her facies was suggestive of possible convulsions. Urine of January 13, shows bloody color, acid reaction, specific gravity 1013, ⅛ to ¼ per cent. of albumen. Sediment has considerable blood normal and abnormal. Much vaginal epithelium, small round cells, occasional uric acid crystals, rare hyaline, fine, brown and fibrous casts.

January 15. At 2.30 A.M. miscarried and living fœtus born. Placenta intact.

At 9 A.M. the morning of the premature birth the patient was seen by Dr. Greenwood, and the eyes examined as thoroughly as possible considering the patient's condition.

O. U. V. = Counting of fingers at two feet, limited to the lower half of the fields of vision. Examination of the fundi shows a well-marked neuro-retinitis, with exudates around the discs and maculæ, and hemorrhages and pigment deposits scattered throughout the retinæ. In each eye there was a nearly symmetrical detachment of the lower quadrant of the retina. These detachments were sector-shaped, with their apices just

below the discs, that in the right eye being ½-disc diameter below, and the one in the left eye 1-disc diameter below the disc. They presented the usual billowy appearance of retinal detachment, and spread out until lost to view near the ora serrata. The writer, on noting their symmetery, and particularly their position in that portion of the eye most dependent, concluded that they were due to œdematous fluid poured out by the choroidal vessel and gravitating to the dependant portion. (On looking up similar cases the writer finds this theory of their cause advocated by Hann and Knaggs in a case reported by them.[1]) Advised treatment by pilocarpine sweats and K. I.

January 29. The fundi were again examined and no detachments found, the only indication of them being a slight haziness of those portions of retina previously detached. There were no fresh hemorrhages, and most of the old ones were absorbed and replaced by pigment deposits. Discs show clearly, though there is still some exudate about them and the maculæ.

By rough testing the fields are normal in extent. V = fingers at four feet.

Examined April 10, 1902:

O. D. V. = movements of fingers.
O. S. V. = 20–70.
O. D. disc white and atrophic, with small arteries, some being reduced to mere threads. The retina is studded with small pigment deposits.
O. S. shows the same fundus changes, with much less atrophy of the disc.

This post-neuritic atrophy, as well as the symmetrical detachments, indicates the severity of the neuro-retinitis with its œdema of the choroid and nerve sheath, and brings to mind the necessity of inducing labor to prevent blindness in similar conditions.

The second case, Miss D., was admitted to the Boston City Hospital, on the service of Dr. George Haven, on February 15, 1902, with the following past history: Menstruation began at

[1] Lancet, May 18, 1901.

12 years, and has always been normal and regular. Three months ago she began to have pains in the lower quadrant. Last week was bothered, while working, by blurring of sight; no chills or vomiting; she admits coitus. Had frequent micturition during past month, appetite variable. Examination showed a well developed but rather slightly built woman, 30 years old. Examination of her heart showed the dulness extending to the second rib on left, with harsh breathing. At third interspace there is a soft, blowing murmur, systolic in time, not heard at the apex. Pulmonic second accentuated. Uterus felt to be enlarged to a point above the umbilicus; superficial veins and tubercles of Montgomery enlarged. Examination of urine made at the time revealed the following condition: Amt. 1014 c.c. sp. gr., 1014 alb., ¼–⅛ sed., frequent hyaline and fine granular casts, a little normal blood, and a few small round cells.

February 16. Cervix is soft, uterus is enlarged to the size of a six months pregnancy. Patient complains of blurring of the sight. She was kept surrounded with hot water bottles, and was heavily covered, but even so the perspiration was sluggish, and she complains a great deal of headache.

February 19. The condition of her eyes was so alarming that Dr. Williams was asked to see the case in consultation. He found the conditions as follows: Both pupils were dilated moderately and equally, vision in each eye was equal to movements of the hands, one foot from the eye. In the fundus of the left eye there was a well marked hemorrhagic retinitis, with a moderate involvement of the optic disc. Just below the macula was a small separation of the retina extending in a horizontal direction towards the disc, and about three disc diameters wide. Small and large hemorrhages covered the entire retina, and there were numerous hemorrhages into the Corpus Vitreum.

In the right eye the same appearances were seen, but there the separation of the retina was larger (about four disc diameters wide), and situated in the same position below the macula. The hemorrhages in the retina and in the Corpus Vitreum were the same as in the left eye.

Upon the results of this examination Dr. Williams advised

an immediate delivery of the fœtus. From that day until February 25 the patient's general condition rapidly grew worse, and on that day the cervix was dilated under ether and a six months fœtus was delivered.

During the following week the patient showed symptoms which were suspicious of maman involvement of the meninges. An examination of the urine made on March 2, showed: Amt. about 20 oz., sp. gr. 1021 alb., ¼ sed., considerable normal blood and a little pus, occasional round highly-refracting cells, many small round cells, rare hyaline and fine granular casts, fat and renal cells adherent.

After March 4 the patient's mental condition improved steadily, so that on March 28 a second examination of her eyes was made. The separation of the retinæ had entirely disappeared, the hemorrhages over the greater part of each retina were still present. The papillæ had begun to look slightly white. At that time the vision in each eye was $\frac{1}{200}$.

The third examination of the eyes was made on April 7. The conditions were as above stated, only the atrophy of each optic disc had progressed considerably, but the vision in each eye had remained the same ($\frac{1}{200}$).

From this time till April 12 the patient's condition gradually improved, but she still complained of blurring of the sight and of almost constant headache. She had a good appetite and slept well.

At the time of discharge on April 12, an examination of the urine showed the condition to be as follows: Amt., about normal; sp. gr. 1011 alb. trace, sed.; frequent squamous cells; rare hyaline and fine granular casts; very rare abnormal blood and also rare renal cells.

In looking up the literature of cases of bilateral retinal detachment, we have been able to collect 11 published cases, a brief synopsis of which follows:

Case 1. By Helbron. — Patient, age 23, eight months pregnant December 27, V. O. U. = hand movements. Retinitis alb., with ablatio retinæ O. U. (Lower half of each retina detached.) Detachments did not float, but looked as if pushed forward by tumors. December 31, artificial birth of dead fœtus. January 10, Neuro-retinitis gone, and detachments O. S. almost completely healed, and O. D. still slightly detached in periphery. V. O. U. 1-4 to 1-3.

February 9. V. O. D. + 50 = 1-2. Reads fine print.
V. O. S. + 50 = 1.0.
F. V. O. U. = Normal. Several months later V. O. D. and O. S. normal.

Case 2. By Brecht. — Patient, age 28, six months pregnant. Retinitis alb. with ablatio retinæ O. U. V. O. D. fingers one foot, V. O. S. fingers $2\frac{1}{2}$ feet. Fourteen days later spontaneous abortion. One month later detachments were gone and retinitis subsiding. Four months later V. O. D. = $\frac{15}{100}$, V. O. S. $\frac{15}{20}$, which did not improve later.

Case 3. By Schweigger. — Patient, age 24. Has had chronic nephritis since attack of diphtheria three years ago. Eight months pregnant. Since one year ago decreased V. O. U. Fundi show an advanced stage of retinitis alb. with ablatic retinæ O. U. (The lower half of each retina being detached.) V. O. D. and O. S. = fingers at one foot. Abortion refused and child born at full term. Two years later complete healing of ablatio retinæ. Perivasculitis of retinal vessels with disseminated choroiditis O. U. vision as above.

Case 4. By Lutz. — Patient, age 25. Had nephritis of long standing. Three months pregnant. Retinitis alb. with albatio retinæ O. U. (Lower half of each retina detached.) Ten days later separations healed; single irregularly running vessel seen in periphery.
V. O. D. + 2.0 = $\frac{5}{36}$ — $\frac{5}{24}$.
V. O. S. + 2.0 = $\frac{5}{24}$ — $\frac{5}{18}$.
Eight days later V. O. D. and O. S. = $\frac{5}{9}$. Twenty-six days after first examination, spontaneous birth of purulent fœtus. One week later no signs of fresh retinitis or albatio retinæ O. U.

Case 5. By Wadsworth. — Patient, age 27. Seven and a half months pregnant. Retinitis alb., with ablatio retinæ O. U. O. D. general separation with thin layer of fluid behind it, more at equator. Retina not quite transparent, with faint gray lines. Forea showed redder oval (not so red as normal color). O. S. separation of less degree with same appearance in macula. V. O. U. not taken. Nine days later artificial delivery. V. O. U. 60 hours after birth = movements of hands in shaded room. Fourteen days later separation O. S. gone and O. D. only slight amount beneath. Two months later V. O. D. = Jæger 13 at 6 — 8″. V. O. S. = Jæger — 5 at 6 — 8″.

Case 6. By Adamück. — Patient, age 30. Eight months pregnant. Retinitis alb. with ablatio retinæ O. U. totalis, and one week completely disappeared with V. O. U. = $\frac{1}{3}$. The retinal detachments were funnel-shaped. A few days after premature delivery only slight detachments of lower parts of each retina were found.

Case 7. By Silex. — Retinitis alb. with ablatio O. U. Retinæ became reattached, but on account of atrophy of optic nerve with chorio-retinitis V. = $\frac{1}{5}$ finally.

Case 8. By Flemming.—Patient, age 30. One day previous amaurosis came with an attack of ecclampsia. Retinitis alb. with ablatio retinæ O. U. Artificial delivery of dead fœtus. Blindness lasted eight days, then V. gradually improved. Fourteen days later ablatio retinæ healed. V. O. D. = $\frac{1}{4}$ O. S. = $\frac{1}{4}$–$\frac{1}{3}$

Case 9. By Guende. — Patient, age 32. Three months pregnant. Retinitis alb. with ablatio retinæ. (The lower half of each retina detached.) Artificial delivery. A few days later V. = light perception. Four years later V. = $\frac{6}{10}$ and fundi normal. Second pregnancy began three months after first confinement, but showed no ocular signs in spite of albumen in urine, and ran an entirely normal course.

Case 10. By Kraskowsky. — Can be mentioned by title only. Retinitis alb. with ablatio retinæ O. U.

Case 11. By Hann and Knaggs.— Patient, age 21. Primipara November 25, 1900. Labor began twenty-four hours before Hann and Knaggs examined her. Semi-comatose, and complained of inability to see three hours before artificial delivery. Remained semi-comatose the next day. Ophthalmoscopic examination made November 27. Ablatio retinæ O. U. lower $\frac{1}{3}$; bilobed by depression in middle. Each disc surrounded by œdematous pallor. No test of V. December 22, the second examination showed no separation. In O. D. and O. S. it was questionable if retinæ were reattached near ora serrata below. Discs O. U. swollen and indistinct, all over each retina irregularly distributed pigment granule spots and patches (the latter in macula of O. D. confluent).
V. O. D. + 1.50 = $\frac{6}{15}$.
V. O. S. + 1.50 = $\frac{6}{12}$. (one letter.)

Perimeter showed V. F. contracted symmetrically in upper hemispheres. January 8, 1901, V. O. D. $\frac{6}{9}$ (two letters), V. O. S. $\frac{6}{9}$ (several letters). No improvement with glasses. January 9, 1901, ophthalmoscopic examination shows no traces of separations V. F. as above.

Helborn, in describing his case,[1] gives a synopsis of the above cases with the exception of the one by Hann and Knaggs. He strongly advises the induction of labor in all cases of retinitis alb. in pregnancy where there is detachment of the retina.

In their case, Hann and Knaggs suggest that impeded venous circulation, resulting from the straining which occurred during labor, was the exciting cause of the retinal detachments.

Their case would seem to support this, but in the other cases collected here, where a record is made as to the time of discovery, including our second case, the retinal detachment occurred before any such exciting cause existed. In the first case the detachments were discovered shortly after delivery, but the history would indicate their being present earlier.

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[1] Berliner Klinische Wockenschrift, January 27 and February 3, 1902.

The fact that, in six of the cases tabulated, together with the first one reported here, the detachments were in, or largely in, the lower part of the retina, would seem to support the theory of the gravitation of œdematous fluid behind the retina, brought forward by Hann and Knaggs.

The occurrence of two cases coming so close together at the City Hospital would suggest to the writers that such cases must be more frequent than would be seemingly indicated by the paucity of published cases, and leads us to urge obstetricians and general practitioners to keep the possibility of such cases in mind, with a view to prevent, by a timely induction of labor, the great or entire loss of useful sight, which may result if such cases go to full term.